May 1990
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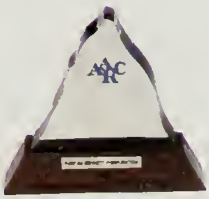

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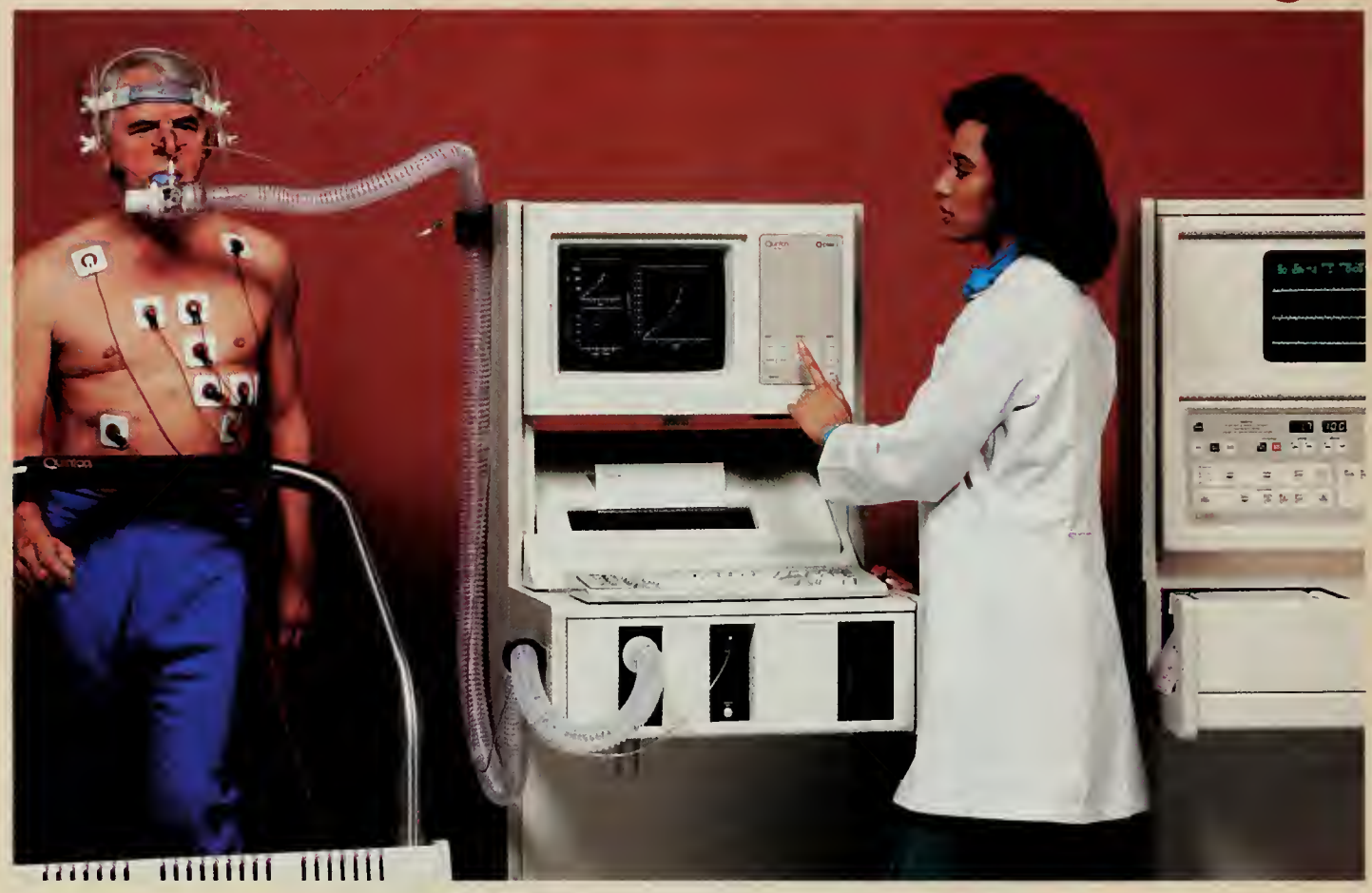

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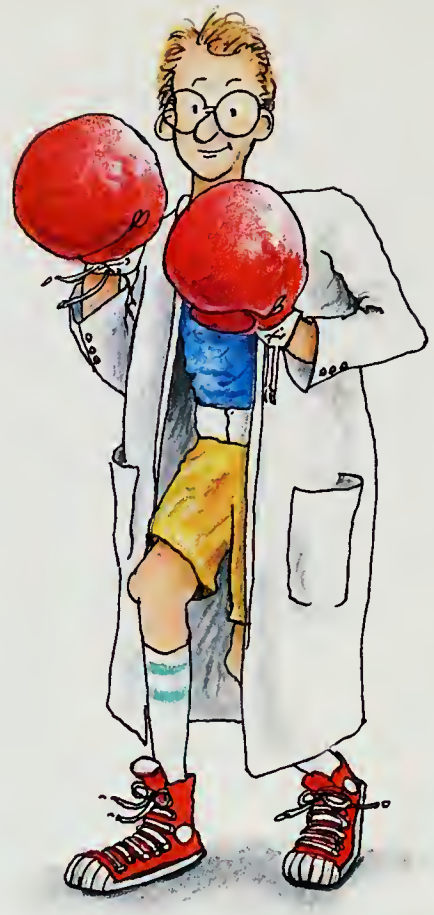

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Long-Term Treatment of Chronic Bronchitis with Positive Expiratory Pressure Mask and Chest Physiotherapy—EF Christensen, T Nedergaard, R Dahl. Chest 1990;97:645.

Long-term treatment of chronic bronchitis with chest physiotherapy with or without positive expiratory pressure (PEP) by mask was studied in 43 patients randomly allocated to PEP treatment (PEP group, 20 patients) and conventional chest physiotherapy (control group, 23 patients). After instruction, the treatments were self-administered twice daily for 12 months (34 patients) and 5 months (9 patients). Twice weekly, patients filled in a diary concerning symptoms. The PEP group had significantly less cough and less mucus production. The number of acute exacerbations were calculated from the diaries and were lower in the PEP group compared to the control group, and 85% of the patients in the PEP group were free from acute exacerbations versus 48% in the control group. The PEP group also used less antibiotics and mucolytics. The PEP group had a small increase in FEV_1 of mean 62 mL compared to a small decrease of 43 mL for the control group. Treatment with a simple PEP device can reduce morbidity in patients with chronic bronchitis and may preserve lung function from a more rapid decline.

Physical Dependence and Attributions of Addiction among Cigarette Smokers—DS Martin. Addictive Behaviors 1990;15:69.

Examination of the attitudes and attributions of cigarette smokers has differentiated smokers who believe their behavior is a "sickness" from those who believe they are "hooked." Among other things, the hooked smoker, more than the sick one, believes they are addicted and their chances of stopping smoking are poor. If there is a causal association between the attribution of addiction and perceived prospects of change, as this suggests, it could mean treatment and preventative programs stressing the addictive nature of cigarettes may be counterproductive. However, the present study, using a survey of 105 male and female smokers from the general population, suggests the attribution of addiction is related to smokers' estimates of their chances of stopping only through a common association that each of these measures has with actual (not necessarily perceived) physical dependence. Caution is needed in the application of cognitive research when related physical measures have not been included in the research design.

Analysis of Breathing Patterns in a Prospective Population of Term Infants Does Not Predict Susceptibility to Sudden Infant Death Syndrome—TB Waggener, DP Southall, LA Scott. Pediatr Res 1990;27:113.

Oscillatory patterns in ventilation have been seen in term and premature infants and are indicative of the stability of the respiratory blood gas feedback control system. Apneas are related to these patterns and apnea duration is correlated with pattern characteristics. In our study breathing patterns were analyzed in recordings from 10 term infants who subsequently died of sudden infant death syndrome (SIDS) and 10 control infants matched for birthweight, gestational age, and postnatal age. Subjects were drawn from a prospectively studied population of 9,856 infants. Breath-by-breath minute ventilation was estimated in each of these 24-h recordings and oscillatory patterns were detected using a comb of digital bandpass filters. Confidence limits on the filter output and a bad data flag for rejection of data during gross body movements or crying ensured that only significant patterns in ventilation were evaluated. Pattern prevalence and amplitude were compared in three frequency regimes: 6- to 87-s cycle times, 6- to 28-s cycle times, and 28- to 87-s cycle times. There was no significant difference between the SIDS and the control infants in any of these pattern comparisons (paired t and Wilcoxon paired rank sum tests, $p < 0.05$). In light of the normal breathing patterns found in the SIDS infants, it is unlikely that susceptibility to SIDS is distinguished, at the time of these recordings, by instability of the respiratory blood gas feedback control system.

Hypoxic Injury to Developing Glial Cells: Protective Effect of High Glucose—DJ Callahan, MJ Engle, JJ Volpe. Pediatr Res 1990;27:186.

Hypoxic injury to differentiating glial cells is a critical event in the development of periventricular leukomalacia, the major hypoxic-ischemic lesion of the premature infant. This study has addressed the effects of hypoxia on differentiating glial cells, primarily astrocytes. Primary cultures of dissociated newborn rat brain, which are composed predominantly of differentiating astroglia, were used. Efflux of lactate dehydrogenase, an enzyme enriched in astroglia, was used to quantitate cellular injury. Three major findings are reported. First, differentiating astrocytes were resistant to hypoxic injury for many hours, although by 24 h of hypoxia severe cellular injury (lactate dehydrogenase efflux of 86% of total and morphologic changes) was obvious. Second, increase of glucose in the culture medium from the approximately physiological concentration of 5.6 to 15 mmol had

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a marked protective effect vs hypoxia, ie, lactate dehydrogenase efflux was totally prevented during 24 h of hypoxia in 15 mmol glucose. Third, the protective effect of high glucose appeared to be related to increased utilization by glycolysis, because there was a direct correlation between the resistance to hypoxic cellular injury and the amount of lactate generated and of glucose consumed by the cells. Thus, the cells with the lowest lactate dehydrogenase efflux (and highest glucose supplementations) had medium lactate concentrations as high as 32-36 mmol. These concentrations of lactate are approximately double the reported threshold concentration of lactate considered to produce cellular necrosis in *in vivo* models of hypoxic injury, primarily in mature animals. The data raise the possibility that hypoxic injury to differentiating glia can be prevented or ameliorated by increase in glucose availability.

A 62-Dose, 6-Month Therapy for Pulmonary and Extrapulmonary Tuberculosis: A Twice-Weekly, Directly Observed, and Cost-Effective Regimen—DL Cohn, BJ Catlin, KL Peterson, FN Judson, JA Sbarbaro. Ann Intern Med 1990;112:407.

Study Objective: To evaluate the efficacy and toxicity of a 62-dose, 4-drug, 6-month, and directly observed regimen for treatment of pulmonary and extrapulmonary tuberculosis.

Design: An open, nonblinded clinical trial, with intended follow-up of patients for 36 months after the completion of therapy.

Setting: A metropolitan tuberculosis clinic in a public health department.

Patients: From March 1981 through April 1989, we enrolled 160 patients with suspected or known tuberculosis; 35 of these patients were excluded from the analysis.

Interventions: Isoniazid, rifampin, pyrazinamide, and streptomycin were administered daily for 2 weeks; these drugs were then given in higher doses twice weekly for 6 weeks, followed by isoniazid and rifampin twice weekly for 18 weeks. A total of 62 doses were administered, and all therapy was directly observed by a nurse or an outreach worker.

Measurements and Main Results: Of the 125 evaluable patients, 101 (81%) had pulmonary tuberculosis, 7 (6%) had both pulmonary and extrapulmonary involvement, and 17 (13%) had extrapulmonary disease only. Seventy-one (57%) patients had a history of recent alcoholism. There were two relapses (1.6% $\pm$ 2.2%), occurring 6 and 56 months after the completion of therapy. The time at which sputum samples became culture negative in pulmonary patients ranged from 1 to 19 weeks (median, 4.6 weeks); 40% $\pm$ 9.6% of patients were culture-negative after 4 weeks of therapy, 75% $\pm$ 8.5% after 8 weeks, 94% $\pm$ 4.7% after 12 weeks, 97% $\pm$ 3.3% after 16 weeks, and 100% after 20 weeks. Adverse drug reactions included hyperuricemia ($>$ 178 μmol/L [3 mg/dL] above normal) secondary to pyrazinamide in 80 patients (64%), twofold or greater elevations of aspartate aminotransferase in 21 patients (17%), 1.5-fold or greater elevations of alkaline phosphatase in 33 patients (27%), cutaneous abnormalities in 8 patients (6%), nausea in five patients (4%), and dizziness in 1 patient (1%).

Conclusions: This 62-dose, largely twice-weekly tuberculosis treatment regimen is efficacious and relatively nontoxic and is especially useful for patients in whom directly observed therapy is indicated.

Colorimetric End-Tidal Carbon Dioxide Monitoring for Tracheal Intubation—JS Goldberg, PR Rawle, JL Zehnder, RN Sladen. Anesth Analg 1990;70:191.

We evaluated a colorimetric end-tidal carbon dioxide ($etCO_2$) detector (FEF end-tidal carbon dioxide detector, Fenem, New York NY) during 62 intubations in anesthetized patients who were hemodynamically stable. The intubations were performed during a drill that simulates difficult tracheal intubation and therefore is associated with an increased risk of esophageal intubation. Each intubation attempt was monitored by two anesthesiologists and a research assistant who together used chest auscultation, colorimetric $etCO_2$, and capnography to confirm tracheal intubation and detect esophageal intubation. The reliability of the monitors was compared with capnography. Colorimetric $etCO_2$ confirmed tracheal intubations and detected esophageal intubations 100% of the time, as judged by capnography. There were no false-positive or false-negative decisions based on endotracheal tube position; however, one equivocal color change occurred, which was caused by failure to inflate the endotracheal tube cuff. Colorimetric $etCO_2$ monitoring confirmed tracheal intubation more rapidly than did chest auscultation ($p < 0.001$) or capnography ($p < 0.05$), and detected esophageal intubation more rapidly than did chest auscultation ($p < 0.05$) and as rapidly as capnography did. Confirmation of tracheal intubation was achieved earlier than detection of esophageal intubation with all three monitors ($p < 0.05$). We conclude that colorimetric $etCO_2$ monitoring is a safe, reliable, rapid, simple, and portable method for determining endotracheal tube position for patients who are hemodynamically stable and should be recommended where capnography is not available.

USPHS Tuberculosis Short-Course Chemotherapy Trial 21: Effectiveness, Toxicity, and Acceptability—CL Combs, RJ O'Brien, LJ Geiter. Ann Intern Med 1990;112:397.

Study Objective: To determine the effectiveness, toxicity, and accepta-

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WARNINGS Excessive use of adrenergic aerosols is potentially dangerous. Fatalities have been reported following excessive use of Alupent® (metaproterenol sulfate USP) as with other sympathomimetic inhalation preparations, and the exact cause is unknown. Cardiac arrest was noted in several cases. Paradoxical bronchoconstriction with repeated excessive administration has been reported with sympathomimetic agents. Therefore, it is possible that this phenomenon could occur with Alupent. Patients should be advised to contact their physician in the event that they do not respond to their usual dose of a sympathomimetic amine aerosol

PRECAUTIONS Because Alupent® (metaproterenol sulfate USP) is a sympathomimetic drug, it should be used with great caution in patients with hypertension, coronary artery disease, congestive heart failure, hyperthyroidism or diabetes, or when there is sensitivity to sympathomimetic amines.
Information for Patients Extreme care must be exercised with respect to the administration of additional sympathomimetic agents. A sufficient interval of time should elapse prior to administration of another sympathomimetic agent
Carcinogenesis Long-term studies in mice and rats to evaluate the oral carcinogenic potential of metaproterenol sulfate have not been completed. Studies of metaproterenol sulfate have not been conducted to determine mutagenic potential or effect on fertility
Pregnancy *Teratogenic Effects. Pregnancy Category C.* Alupent has been shown to be teratogenic and embryocidal in rabbits when given orally in doses 620 times the human inhalation dose and 62 times the human oral dose. There are no adequate and well-controlled studies in pregnant women. Alupent should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.
Oral reproduction studies in mice, rats and rabbits showed no teratogenic or embryocidal effects at 50 mg/kg, corresponding to 310 times the human inhalation dose and 31 times the human oral dose. Teratogenic effects in the rabbit included skeletal abnormalities and hydrocephalus with bone separation.
Nursing Mothers It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk, caution should be exercised when Alupent is administered to a nursing woman.
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ADVERSE REACTIONS Adverse reactions are similar to those noted with other sympathomimetic agents. Adverse reactions such as tachycardia, hypertension, palpitations, nervousness, tremor, nausea and vomiting have been reported.
The most frequent adverse reactions to Alupent® (metaproterenol sulfate USP) Inhalation Solution are nervousness and tachycardia which occur in about 1 in 7 patients, tremor which occurs in about 1 in 20 patients and nausea which occurs in about 1 in 50 patients. Less frequent adverse reactions are hypertension, palpitations, vomiting and bad taste which occur in approximately 1 in 300 patients.

HOW SUPPLIED ***Inhalation Aerosol:*** Each canister of Alupent® (metaproterenol sulfate USP) Inhalation Aerosol contains 225 mg of metaproterenol sulfate as a micronized powder in inert propellants. Alupent Inhalation Aerosol with mouthpiece (15 ml). Alupent Inhalation Aerosol refill (15 ml).
Store below 77°F (25°C). Avoid excessive humidity.
Inhalation Solution: Alupent Inhalation Solution is supplied as a 5% solution in bottles of 10 ml or 30 ml with accompanying calibrated dropper.
Store below 77°F (25°C). Protect from light. Do not use the solution if it is brown or has a precipitate.
Alupent Inhalation Solution Unit-dose Vial is supplied as a 0.4% or 0.6% clear colorless or nearly colorless solution containing 2.5 ml with 25 vials per box. Store below 77°F (25°C). Protect from light. Do not use the solution if it is brown or has a precipitate.
Syrup: Alupent is available as a cherry-flavored syrup, 10 mg per teaspoonful (5 ml), in 16 fl. oz. bottles. Store below 86°F (30°C). Protect from light.
Tablets: Alupent is supplied in two dosage strengths as scored, round white tablets in bottles of 100. Tablets of 10 mg coded BI/74. Tablets of 20 mg coded BI/72.
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bility of a 6-month antituberculous regimen compared with a 9-month regimen.

Design: A nonblinded, unbalanced, randomized, multicenter clinical trial.

Setting: Twenty-two tuberculosis clinics in public health departments and hospitals in the United States.

Patients: Patients were eligible if *Mycobacterium tuberculosis*, isolated from sputum cultures, was susceptible to study drugs. Of 1451 patients enrolled, 75% (617 of 823) assigned to the 6-month regimen and 71% (445 of 628) assigned to the 9-month regimen were eligible.

Interventions: Patients took self-administered isoniazid and rifampin daily for 24 weeks (6-month regimen) or 36 weeks (9-month regimen). In addition, patients assigned to the 6-month regimen took self-administered pyrazinamide daily during the first 8 weeks.

Results: Patients on the 6-month regimen converted more rapidly than patients on the 9-month regimen (94.6% compared with 89.9% after 16 weeks of therapy, with a difference of 4.7% [95% CI, 0.7% to 8.7%]); had similar rates of adverse drug reactions (7.7% compared with 6.4%, with a difference of 1.3% [95% CI, 0.0% to 4.6%]); had lower noncompliance rates (16.8% compared with 29.2%, with a difference of 12.4% [95% CI, 6.8% to 18.0%]); and had similar relapse rates 96 weeks after completing therapy (3.5% compared with 2.8%, with a difference of 0.7% [95% CI, 0.0% to 3.9%]). A significantly greater proportion of patients assigned to the 6-month regimen successfully completed therapy (61.4% compared with 50.6%; $\chi^2 = 11.976$).

Conclusions: Our results suggest that this 6-month regimen is similar in effectiveness, toxicity, and acceptability to the 9-month regimen for treating pulmonary tuberculosis.

Reference Equations Used To Predict Pulmonary Function: Survey at Institutions with Respiratory Disease Training Programs in the United States and Canada—AJ Ghio, RO Crapo, CG Elliott. Chest 1990;97:400.

Adult respiratory disease training programs in the United States and Canada were surveyed to determine which reference equations were used to predict normal pulmonary function and how ethnic differences were approached. Replies from 139 of the 180 (77.2%) institutions surveyed were received and evaluated. Surprisingly few studies account for most of the equations in use: three studies account for 85% of the spirometric equations, two for 83% of the lung volume equations and five for 84% of the diffusing capacity equations. Although there are no definite data, the form of many of the replies suggests that equipment default settings may influence the selection process. Of those responding to the ethnic difference question, 53% of institutions applied no correction for ethnic differences. There was no consistent pattern to the method of correction among those who did.

Performance Evaluation of Contemporary Spirometers—SB Nelson, RM Gardner, RO Crapo, RL Jensen. Chest 1990;97:288.

A comprehensive evaluation of 62 spirometers from 37 different sources was performed using a two-part protocol: calibrated syringe, and dynamic waveform testing. All testing was done with ambient air. Calibrated syringe testing examined the ability of the spirometers to accurately measure the output of a 3-L calibrating syringe under varying conditions. The accuracy, FVC volume linearity, and stability of each spirometer was determined from these data. All but 5 of 42 spirometers accurately measured a 3-L calibrating syringe to within ± 3%. Dynamic waveform testing consisted of introducing 24 standard waveforms into the spirometer from a computer-controlled air pump. The values of FVC, FEV_1, and FEF_{25-75} were compared to the actual values for each waveform to determine a performance rating. Only 35 (56.5%) of the spirometers performed acceptably when measuring the 24 standard waveforms. Nine (14.5%) were marginal and 18 (29.0%) were unacceptable. Fifty-nine (95%) of the 62 spirometers were computerized. Software errors were found in 25% of the computerized systems evaluated. Although using a 3-L syringe for quality control purposes is essential, simple testing of spirometers with a 3-L calibrating syringe for validation purposes was inadequate to assess spirometer performance when compared to dynamic waveform testing. Dynamic waveform testing is essential to accurately measure and validate acceptability of spirometer system performance.

Smell and Taste Function in Subjects with Chronic Obstructive Pulmonary Disease: Effect of Long-Term Oxygen via Nasal Cannulas—NA Dewan, W Bell, J Moore, B Anderson, W Kirchain, WJ O'Donohue Jr. Chest 1990 97:595.

It was hypothesized that subjects with chronic obstructive pulmonary disease (COPD) receiving long-term oxygen via nasal cannulas have an impaired sense of smell and/or taste. To objectively evaluate the sense of smell and taste, this study used the University of Pennsylvania Identification Test (UPSIT), a 40-item "scratch-n-sniff" test and a 20-item taste test using the four basic taste sensations of sweet, salt, sour, and bitter. Twenty subjects (15 male, 5 female) with severe COPD receiving long-term oxygen therapy (group 1), and an equal number of age- and sex-matched subjects with COPD not receiving oxygen therapy (group 2), and a healthy control group (group 3) were studied. Twelve subjects (seven male, five female) from group 1 subsequently underwent transtracheal oxygen catheter installation. Mean ± SD for the basic smell test was significantly greater in group 3 (35.35

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± 3.58) as compared with group 1 (27.70 ± 6.07) or group 2 (31.10 ± 4.95) ($p < 0.005$). The difference between group 1 and 2 was not significant ($p = 0.066$). However, when adjusted for pack years of smoking, there were no significant differences between the three groups. Mean ± SD correct responses for the basic taste test were significantly greater in group 3 (15.75 ± 1.81) as compared with group 2 (12.8 ± 2.78) ($p < 0.005$) and group 1 (14.00 ± 2.33) ($p < 0.05$). There was no significant difference between group 1 and 2. The corrected data for taste, adjusted for years since quitting smoking, did not alter the basic differences between the groups. Mean smell and taste test scores were essentially unchanged in 12 subjects after six months of transtracheal oxygen therapy. Long-term oxygen use via nasal cannulas in this group of subjects with COPD did not appear to impair their sense of smell and taste. Smoking had a significant but variable effect on the sense of smell and taste.

Reliability of Pulse Oximetry during Exercise in Pulmonary Patients—PJ Escourrou, MF Delaperche, A Visseaux. Chest 1990;97:635.

To evaluate the reliability of pulse oximetry during exercise, we studied 101 patients primarily with chronic pulmonary diseases. Three devices were used on different patients. Radial arterial blood was sampled at rest and maximal exercise simultaneously to pulse oximetric determination. Measured blood oxygen saturation was significantly different from noninvasive saturation at rest and also at exercise for each device. Nevertheless, changes in pulse oximetry from rest to exercise were significantly correlated with measured saturation for all three devices. Direction of changes in saturation from rest to exercise was correctly evaluated by transcutaneous oximetry in all but six instances where changes were less than 4%. Although measured and transcutaneous saturations are significantly different, we conclude that pulse oximetry reliably estimates changes in arterial saturation between rest and exercise for a clinical purpose. None of the three tested devices was better compared with the others in estimating saturation changes at exercise.

Physiologic Effects and Side Effects of Prostaglandin E_1 in the Adult Respiratory Distress Syndrome—JA Russell, JJ Ronco, PM Dodek. Chest 1990;97:684.

Because PGE_1, previously has been reported to increase survival of patients with ARDS, we evaluated physiologic effects and side effects of PGE_1, in a prospective open-label study of patients with ARDS. Seventeen patients with ARDS who did not have significant renal or hepatic dysfunction received PGE_1 by continuous central venous infusion (30 ng/kg/min). Seventeen control patients with ARDS without renal or hepatic dysfunction who had similar APACHE II and ARDS scores and causes of ARDS did not receive PGE_1. Prostaglandin E_1 significantly decreased the SVRI and oxygen extraction ratio. Concentrations of total and polymorphonuclear leukocytes, but not platelets, increased significantly during PGE_1 infusion, but did not change in control patients. There was no change in the $D_{O_2}I$ and $\dot{V}_{O_2}I$ during the course of the PGE_1 infusion. There were no differences in $D_{O_2}I$ and $\dot{V}_{O_2}I$ during PGE_1 infusion between survivors and nonsurvivors. Prostaglandin E_1 was infused for a mean of 5.9 ± 1.8 days (± SD) and was discontinued on ten occasions in seven patients because of supraventricular dysrhythmias (n = 4), hypotension (n = 3), thrombocytopenia (n = 3), and cardiac arrest (n = 2). Nonsurvivors had PGE_1 discontinued prematurely more frequently than survivors (56% [5/9] vs 25% [2/8], respectively). The prevalence of multiple-system organ failure and the in-hospital mortality of both PGE_1-treated and control patients were not different. Although PGE_1 causes significant systemic vasodilation and possibly decreased intrapulmonary polymorphonuclear leukocyte sequestration, PGE_1 does not influence multiple-system organ failure or mortality of patients with ARDS without renal or hepatic dysfunction.

Changes in Airflow Obstruction and Oxygen Saturation in Response to Exercise and Bronchodilators in Cystic Fibrosis—PI Macfarlane, D Heaf. Pediatr Pulmonol 1990;8:4.

The airway response to exercise and inhaled terbutaline was assessed in 25 patients with cystic fibrosis (CF), seeking evidence for the possible deleterious effects of bronchial muscle relaxation. We postulated that 'early' and 'late' flows, taken from the full maximum expiratory flow volume curve, might move paradoxically in patients with unstable airways. Oxygen saturation was measured continuously; desaturation occurred early in exercise with partial recovery thereafter. This was unrelated to changes in expiratory airflow measurements. Both during and after exercise, and after inhaled bronchodilator, changes in expiratory airflow measurements were strikingly variable. Changes in individual measurements should be interpreted in relationship to the within-subject variability of the test in patients with CF. During exercise, there was a significant increase in mean FEV_1; this was most marked in patients with worst lung function. Two patients (both with severe lung disease) showed paradoxical changes in early and late flows. After exercise, only two patients showed the asthmatic pattern of postexercise bronchoconstriction. After inhaled bronchodilator, the group as a whole showed small but statistically significant increases in expiratory airflow measurements. Those with highest baseline FEV_1 had the greatest bronchodilator response;

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this is the opposite of the pattern observed in asthma. Paradox did not occur after bronchodilators and only one patient showed a significant fall in late expiratory airflow. This pattern of expiratory airflow changes is compatible with the concept of airway instability in which any beneficial effects of bronchial tone reduction are cancelled out by the effects of compression of damaged airways rendered more compliant by loss of bronchial wall tone. We did not observe any clinically important deleterious effects from this mechanism.

Diagnostic Value of Body Plethysmographic Parameters in Healthy and Asthmatic Young Children Is Not Influenced by Breathing Frequency—W Buhr, R Jorres, M Knapp, D Berdel. Pediatr Pulmonol 1990;8:23.

In 16 healthy and 16 asymptomatic asthmatic children (age range 5-8 yr; 8 girls, 24 boys) we studied the influence of breathing frequency on the results and the diagnostic value of body plethysmographic measurements. Airway resistance (R_{aw}), specific airway resistance (SR_{aw}), and thoracic gas volume (TGV) were measured during breathing (or breathing efforts against a closed shutter) at 0.4, 1, and 2 Hz. SR_{aw} was computed by a simplified procedure directly from flow at the mouth vs box volume-curves. The diagnostic value of each parameter was assessed as the percentage of correctly classified healthy and asthmatic subjects by means of discriminant analysis. When frequency was increased from 0.4 to 1 and 2 Hz, mean TGV rose by 5 and 14% in healthy children and by 11 and 21% in asthmatic children, respectively. From 0.4 to 1 Hz, mean R_{aw} decreased by 16% ($p = 0.002$) in healthy children and by 25% ($p = 0.0004$) in asthmatic children. The differences in R_{aw} between both groups decreased with frequency (3.5, 1.8, and 1.5 cm $H_2O \cdot s \cdot L^{-1}$ at 0.4, 1, and 2 Hz, respectively) and those of TGV increased (0.13, 0.21, and 0.23 L). SR_{aw} showed similar frequency characteristics as R_{aw}. As intra-group variability changed in parallel with the differences, the diagnostic value of the parameters remained constant with frequency. Simplified SR_{aw} alone and TGV combined with R_{aw} exhibited no differences in their diagnostic values (81-84% correctly classified). Our results indicate that in young healthy and asthmatic children the diagnostic value of TGV, R_{aw}, and SR_{aw} is not influenced by breathing frequencies (0.4-2 Hz) and that in these children the simplified computation of SR_{aw} can be used without loss of information.

A Controlled Trial of Human Surfactant Replacement Therapy for Severe Respiratory Distress Syndrome in Very Low Birthweight Infants—MJ Lang, RT Hall, NS Reddy, CG Kurth, TA Merritt. J Pediatr 1990;116:295.

In a randomized, controlled study, human surfactant derived from amniotic fluid was administered within 12 hours of birth to infants with severe respiratory distress syndrome who were born at 24 to 32 weeks of gestation weighing $\leqslant$ 1500 g. A second dose of surfactant was given to patients in the treatment group if they met ventilator requirements indicating relapse or lack of response to the initial dose. No significant improvement was observed in mortality rate (9/28 vs 15/31) or incidence of bronchopulmonary dysplasia (5/28 vs 3/31) when surfactant-treated infants were compared with control subjects, although there was a significant reduction in initial respiratory and inspired oxygen requirements and the arterial/alveolar oxygen ratio improved. In addition, there was a significant reduction in pulmonary air leak in treated infants (10/28 vs 20/31; $p < 0.05$). Retreatment was associated with an attenuated ventilatory response and with a higher mortality rate (7/14) than that of infants who did not require a second dose (2/14; $p = 0.05$), indicating a more severe form of disease. Multiple discriminant analysis, including eight independent variables, revealed that increasing birthweight, earlier age at surfactant treatment, and female gender were significantly associated with survival. These data suggest that early surfactant treatment may reduce mortality rates in very low birthweight infants with severe respiratory distress syndrome, as well as reduce ventilator requirements and the incidence of pulmonary air leaks.

Low Sensitivity of the Anion Gap as a Screen To Detect Hyperlactatemia in Critically Ill Patients—TJ Iberti, AB Leibowitz, PJ Papadakos, EP Fischer. Crit Care Med 1990;18:275.

The anion gap is commonly used as a screening test for the presence of lactic acidosis. Analysis of the distribution of anion gaps for 56 adult surgical ICU patients with peak blood lactate levels $\geqslant$ 2.5 mmol/L showed the anion gap to be an insensitive screen for elevated lactate in a critically ill, hospitalized population. All patients (11/11) with peak lactate $\geqslant$ 10 mmol/L had an anion gap $\geqslant$ 16 mmol/L; however, 50% (6/12) of patients with lactates between 5.0 and 9.9 mmol/L and 79% (26/33) of those with lactates between 2.5 and 4.9 mmol/L had anion gaps $<$ 16 mmol/L. Hyperlactatemia was associated with considerable mortality at all levels: 100% among patients with lactate levels $\geqslant$ 10 mmol/L, 75% between 5.0 and 9.9 mmol/L, and 36.4% between 2.5 and 4.9 mmol/L. Acidosis (pH $<$ 7.30) did not significantly alter mortality by lactate level. The observation that, for 57% of patients in this study, an elevated lactate level was not accompanied by an elevated anion gap suggests that hyperlactatemia should be included in the differential diagnosis of non-anion gap acidosis.

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Dose-Related Effects of Cigarette Smoking on Olfactory Function—RE Frye, BS Schwartz, RL Doty. JAMA 1990;263:1233.

Little is known about the influence of cigarette smoking on the ability to smell; previous studies on this topic have led to contradictory findings and have failed to take into account smoking dose and duration. In the present study, the 40-odorant University of Pennsylvania Smell Identification Test was administered to 638 subjects for whom detailed smoking histories were available. Smoking was found to be adversely associated with odor identification ability in a dose-related manner in *both* current and previous cigarette smokers. Among previous smokers, improvement in olfactory function was related to the time elapsed since the cessation of smoking. Logistic regression analysis found current smokers to be nearly twice as likely to evidence an olfactory deficit than persons who have never smoked. Overall, the data suggest that (1) smoking causes long-term but reversible adverse effects on the ability to smell and (2) the failure of some studies to demonstrate smoking effects may be caused by the inclusion of persons with a history of smoking in the nonsmoking groups.

The Older Smoker: Status, Challenges and Opportunities for Intervention—BK Rimer, CT Orleans, MK Keintz, S Cristinzio, L Fleisher. Chest 1990;97:547.

Smoking is a significant cause of morbidity and mortality among older adults. Cessation of smoking benefits older adults almost immediately. Little is known, however, about how older adults quit and how to help them. No smoking cessation programs have been designed for this population. Here we report the findings of a random survey of American Association of Retired Persons members conducted to learn more about older smokers, their smoking and health characteristics, their quitting motivations and experiences, and the role of physicians' advice to quit. We obtained data on 339 current smokers aged 50 to 102. Current smokers were more likely to be heavy, highly addicted smokers. They also reported more smoking-related symptoms and conditions and fewer preventive tests and check-ups than never-smokers or former smokers. Although 44% of smokers were interested in quitting, only 39% reported that they had been advised to stop smoking by their physicians in the previous year. Physicians who treat older patients can have a significant impact on helping them to stop smoking by giving them a strong recommendation to quit and by providing appropriate interventions.

Pathogenesis of Colonization and Infection in a Neonatal Surgical Unit—EM Leonard, HKF van Saene, P Shears, J Walker, PKH Tam. Crit Care Med 1990;18:264.

Nosocomial infection with aerobic gram-negative bacilli is a major cause of morbidity and mortality in neonates. Few prospective studies have been undertaken in neonatal surgical units to investigate colonization and infection rates and the pathogenesis of infection. We prospectively studied 40 infants admitted to a neonatal surgical unit. Ninety-eight percent became colonized in throat/intestine with aerobic gram-negative bacilli. Thirty-five percent developed infections, with wound and surface infections predominant (61%). Ninety-one percent of infections were caused by gram-negative bacilli or yeasts. Severe infections (septicemia, pneumonia, meningitis) occurred in 13% of infants. The mortality rate was 5%. In all infections, the pathogenesis was found to be endogenous, and, in most, three stages were distinguishable. Neonates always acquired potentially pathogenic organisms in throat/intestine (Stage 1) before colonization (Stage 2) and infection (Stage 3) of other systems occurred. Reduction of digestive tract colonization by these potentially pathogenic microorganisms by means of successful selective decontamination may therefore reduce subsequent infection.

The Relationship between Pulmonary Function and Dyspnea in Obstructive Lung Disease—N Wolkove, E Dajczman, A Colacone, H Kreisman. Chest 1989;96:1247.

Increased importance is now being placed on evaluating dyspnea in patients with obstructive lung disease (OLD). We measured breathlessness at rest, using a Borg scale dyspnea index (BSDI) before and after bronchodilator (albuterol [salbutamol] 200μ) in 93 patients with OLD drawn from a larger population undergoing routine spirometry. The median BSDI declined from 3 to 1 before and after bronchodilator, suggesting improvement in dyspnea. However, there was no correlation between initial or post-bronchodilator spirometry and BSDI. The change in FEV_1 similarly did not correlate with the change in BSDI ($r = 0.05$). A large bronchodilator response was usually associated with improvement in dyspnea, but the converse was not observed. Thus, of ten patients with an improvement in BSDI of more than two categories, six had a change in FEV_1 of 0.1 L or less after bronchodilator. Analyzing a subgroup of 65 dyspneic patients with an initial BSDI of 2 or more, revealed the following response groups: those with either a bronchodilator or dyspnea response alone, both together, or neither. Twenty-eight patients (43%) responded both subjectively and objectively. Eleven (17%) had a bronchodilator response only, seventeen (26%) had a dyspnea response only, while nine (14%) had neither measurable response. We conclude

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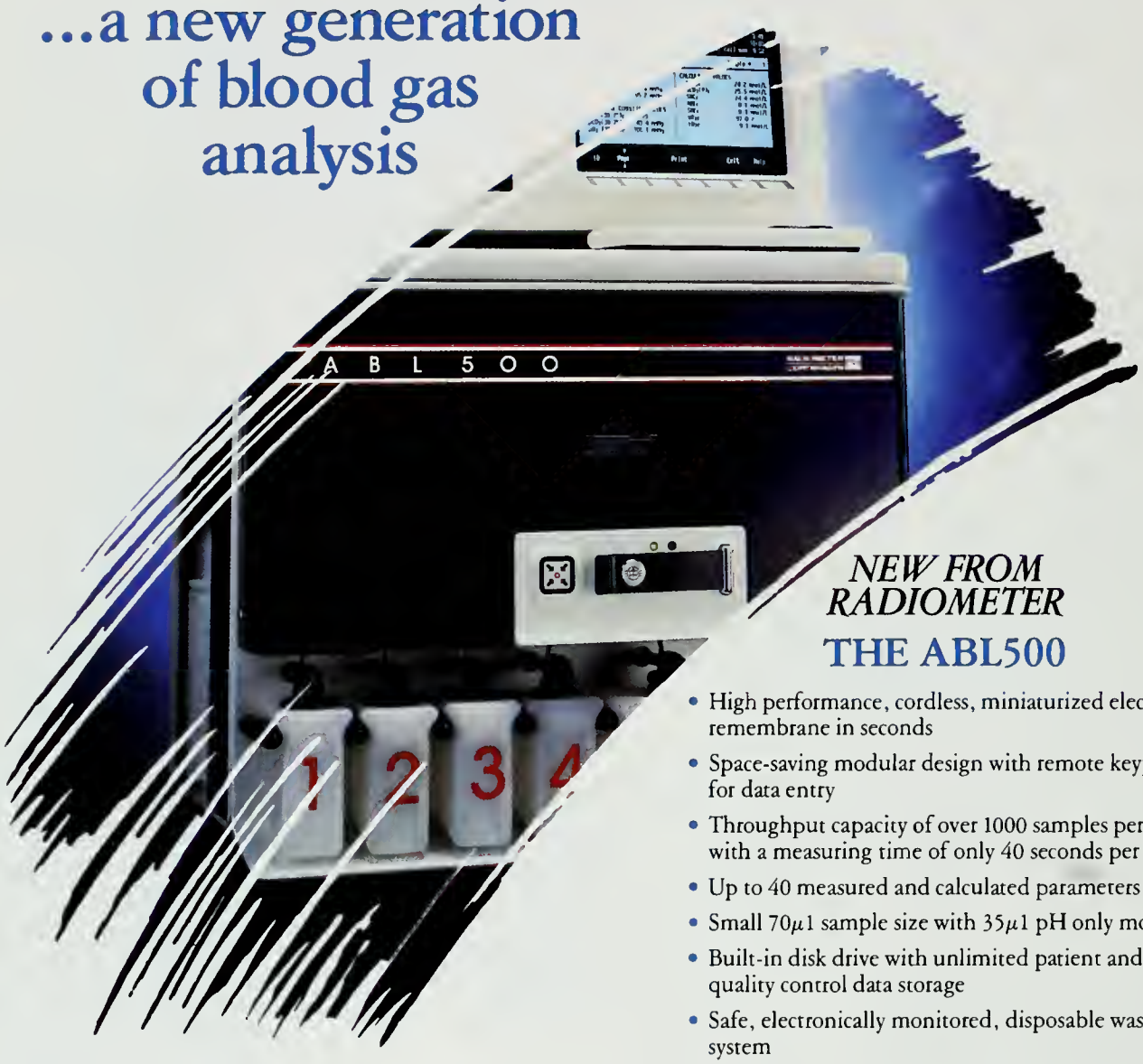

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that dyspnea is poorly correlated with results of routine spirometry in patients with OLD. The use of dyspnea ratings may yield information about bronchodilator responsiveness not appreciated by spirometry alone.

Can Maximal Cardiopulmonary Capacity be Recognized by a Plateau in Oxygen Uptake?—J Myers, D Walsh, N Buchanan, VF Froelicher. Chest 1989;96:1312.

The failure of oxygen uptake to increase with increasing work has been considered a marker of the limits of the cardiopulmonary system for many years. However, the concept has suffered from inconsistencies in definition, criteria, and data sampling, all of which affect the interpretation of the relation between changes in work and oxygen uptake. To evaluate the response and reproducibility of the slope in oxygen uptake at peak exercise, six subjects (mean age, 33 ± 6 years) performed two individualized ramp treadmill tests on separate days. During exercise, oxygen uptake (for a given sample of 30 eight-breath running averages) was regressed with time and the slope was calculated. Maximal oxygen uptake, maximal heart rate and maximal perceived exertion were reproducible from day 1 to day 2 (mean difference, 0.4 ml/kg/min, 1.0 beats per minute, and 0.2 for maximal oxygen uptake, heart rate, and maximal perceived exertion, respectively [not significant]). Considerable variability in the slopes was observed during each test and from day to day. This occurred despite the use of large gas exchange samples, averaging techniques, and constant, consistent changes in external work. A plateau, defined as the slope of an oxygen uptake sample at peak exercise that did not differ significantly from a slope of zero, was not a consistent finding within subjects between days. We conclude that marked variability in the slope of the change in oxygen uptake occurs throughout progressive exercise, despite the use of large samples and a linear change in external work. These findings appear to preclude the determination of a plateau by common definitions.

Variability of Pulmonary Function Tests in Cystic Fibrosis—PJ Cooper, CF Robertson, IL Hudson, PD Phelan. Pediatr Pulmonol 1990;8:16.

The aim of this study was to define the within-subject variability for tests of respiratory function in patients with cystic fibrosis (CF) within the day, from day to day, and from week to week. Twenty-eight patients with CF (aged 9-19 years) and 23 healthy height-matched controls (aged 9-18 years) had measurements made of spirometry, lung volumes, maximal flows at three lung volumes, and maximal inspiratory and expiratory pressures at the mouth. Testing was done on nine occasions, three times within a day, on consecutive days at 1-week intervals. Each individual's variability was summarized both as the within-subject coefficient of variation (WCV) and within-subject standard deviation (WSD). Means of WSD and median WCV are reported for both the patients with CF and normal subjects. The within-subject variability of VC, FEV_1, TLC, RV, and RV/TLC was more appropriately assessed by the use of WSD rather than WCV. The WSDs in the CF group were significantly more variable ($p < 0.005$) than in the normals for VC and FEV_1. WCV best summarized within-subject variation for $FEF_{25-75\%}$, FRC, $\dot{V}_{25}$, $\dot{V}_{50max}$, and $\dot{V}_{75max}$ for which the CF subjects were significantly more variable ($p < 0.005$). Individuals' variability was very consistent, therefore assessment of significant change could be made more accurately by predetermining the variability of that individual, rather than using group data. We stress the importance to consider increased variability from day to day and week to week in the interpretation of change in lung function in patients with CF, and provide reference values for accurate interpretation of serial pulmonary function test results.

Clinical Efficacy and Cost Benefit of Pulse Flow Oxygen in Hospitalized Patients—GR Kerby, WJ O'Donohue, DJ Romberger, FN Hanson, GA Koenig. Chest 1990;97:369.

Pulse flow oxygen administered during early inspiration is a promising approach to oxygen conservation. Previous short-term studies show equivalent arterial P_{O_2}, 55 to 60% oxygen savings, and no reduction of nasal humidity when compared with continuous flow nasal cannula oxygen. This study compares the clinical efficacy of pulse flow and continuous flow oxygen in 100 patients recently hospitalized for diseases requiring O_2 therapy. In an unblinded crossover design, pulse and continuous O_2 were administered alternately during four 5½-hour periods. Oxygen saturation was monitored continuously during the 23-hour study. Mean S_{aO_2} on pulse flow (95.6 ± 2.7%) was clinically the same as continuous flow (95.3 ± 2.6%). Mean S_{aO_2} on pulse flow during the 30 minutes before or after each crossover (95.5 ± 3.3%) was similar to continuous flow during the 30 minutes near crossover (95.3 ± 3.1%). It is concluded that the two delivery systems produce similar levels of S_{aO_2} over the course of a day and night. Analysis of potential cost savings achieved by use of the device for a 350-bed hospital suggests a savings of about $50,000 yearly when accompanied by termination of oxygen humidification.

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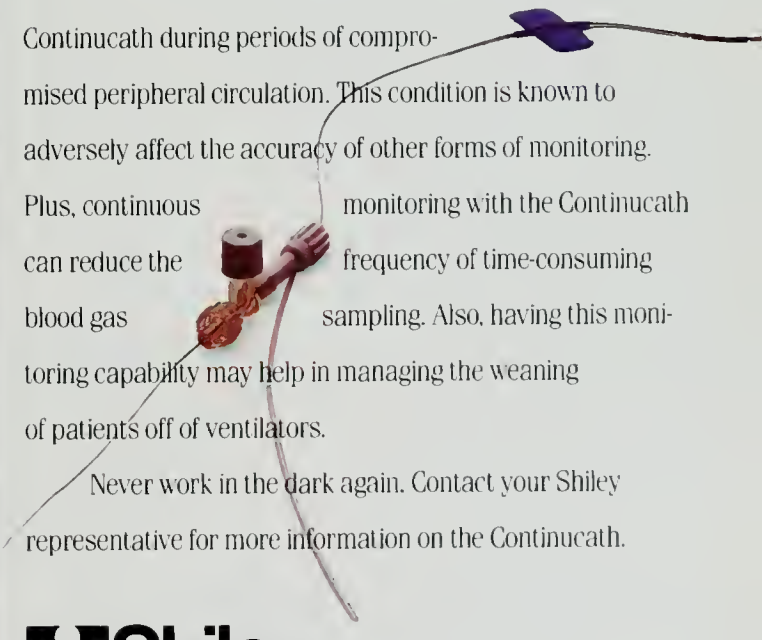

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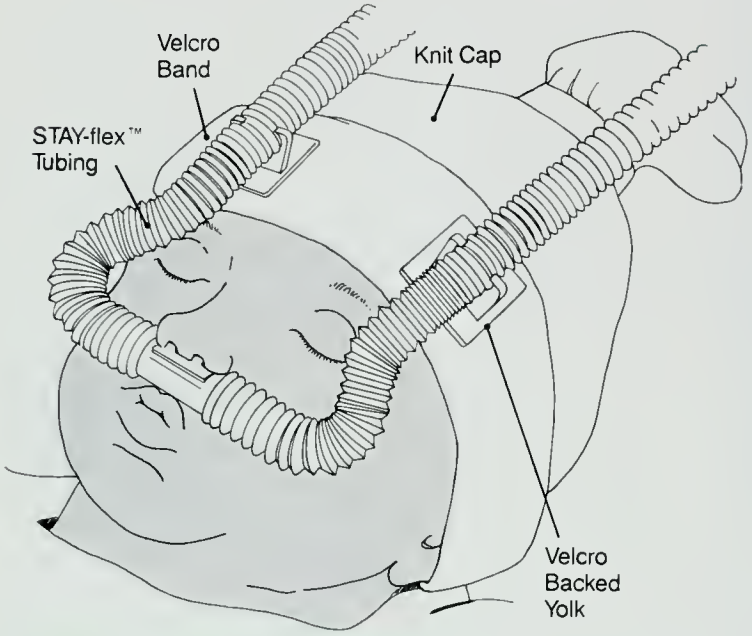

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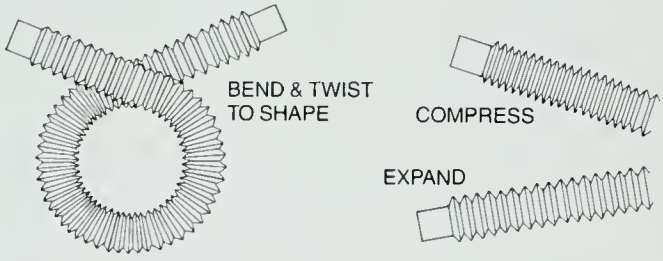

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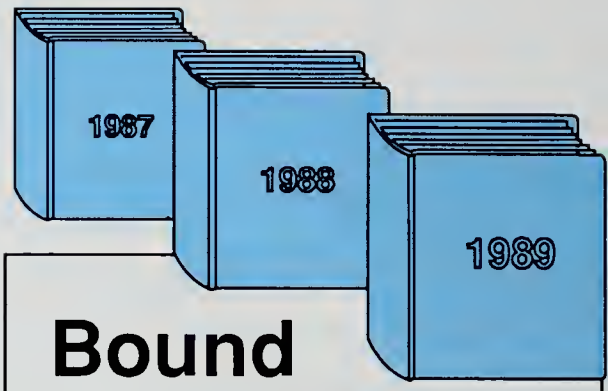

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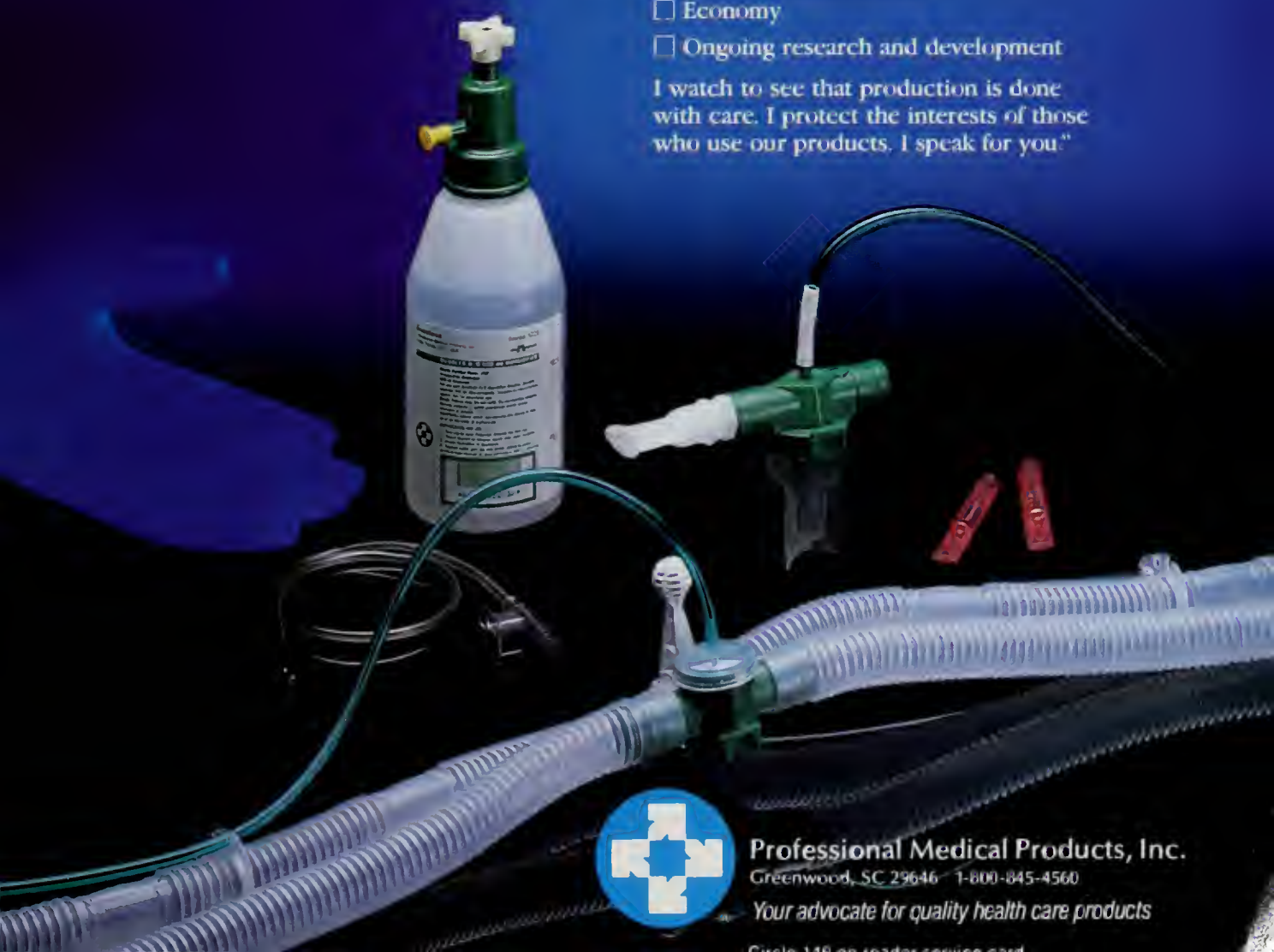

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The Delivery of Aerosolized Bronchodilator to Mechanically Ventilated Intubated Adult Patients

Aerosolized bronchodilators are commonly administered to intubated adult patients, often by use of a small volume nebulizer (SVN). Although interest in the delivery of these medications by metered dose inhaler (MDI) has increased, very little has been published regarding the delivery of aerosolized medications to intubated patients. MacIntyre et al[1] studied radioactive aerosol delivery in 7 mechanically ventilated adult patients, and found that only 2.9 ± 0.7% of the radioactive particles reached the lungs. In another group of 15 mechanically ventilated patients, MacIntyre et al[1] found that aerosolized metaproterenol had no significant effect on lung mechanics. On the other hand, in a study of aerosolized pentamidine in patients with pneumocystosis, Girard et al[2] found greater plasma concentrations of the drug with ventilated than with nonventilated patients.

Zandstra et al[3] found that nebulization of a mucolytic agent resulted in an increase in airway resistance in 10 mechanically ventilated postoperative patients. The effect of the mucolytic agent was blocked by the addition of a bronchodilator (salbutamol, or albuterol) to the aerosol. This suggests that enough of the aerosol was delivered to the lungs to result in physiologic effects. At least two studies[4,5] have shown a decrease in airway resistance in ventilator-dependent infants with bronchopulmonary dysplasia, following the administration of aerosolized bronchodilator.

In an in-vitro model, Ahrens et al[6] showed that the endotracheal tube serves as a formidable barrier to the penetration of therapeutic aerosols into the lower respiratory tract. Using a FanJet nebulizer, they showed that the amount of aerosol delivered into a lung model was dependent on endotracheal tube size and inspiratory flow. Deposition of aerosol in the lung model decreased as endotracheal tube size decreased and as inspiratory flow increased. In that bench study, it was also found that the amount of aerosol delivered into the lung model was increased when a nebulizer that produced aerosol particles with a mass median diameter of 0.54 μ, rather than the FanJet with a particle size of 3.95 μ, was used.

Hughes and Saez[7] used a bench model to evaluate the effect of nebulizer mode and position on the amount of medication available to the mechanically ventilated patient. Using a radioactive aerosol, they found that significantly more medication was available at the Y-piece of the ventilator circuit if the nebulizer was placed at the manifold, and if the nebulizer was operated during inspiration only. It is interesting to note that the least amount of aerosol was available to the patient with the nebulizer placed at the Y-piece and operating continuously—the technique that appears to be the one most commonly used in clinical practice. However, that study evaluated only the amount of aerosol delivered to the endotracheal tube, and did not evaluate the amount of aerosol that passed through the endotracheal tube.

It has been shown that a significant amount of the solution placed into a SVN may be trapped in the nebulizer.[8–10] This decreases the amount of aerosol delivered to the patient. The performance of a SVN is affected by diluent volume, flow, and nebulizer brand. As the result of a bench evaluation of SVN function, my co-workers and I recommend that these nebulizers be used with a 4-mL diluent volume, and be powered with a flow of 8 L/min.[8] Johnson et al[11] have shown that nebulizer brand and flow can affect the deposition and physiologic response to aerosolized bronchodilator delivery.

It is very difficult to evaluate the response of intubated patients to aerosolized bronchodilator. Although spirometry is commonly used to evaluate response in ambulatory patients, the technique is not feasible in intubated mechanically ventilated patients.

Further, the endotracheal tube is a major site of resistance in intubated patients.[12] Thus, the fixed resistance of the endotracheal tube may mask changes in airway resistance in the lungs. Gay et al[13] have suggested that changes in peak airway pressure and auto-PEEP may be useful in the evaluation of bronchodilator response in intubated patients. A number of current-generation mechanical ventilators provide measurements and calculations of lung mechanics that may be useful in the evaluation of bronchodilator response. However, it must be recognized that the measurements of lung mechanics provided by some of these ventilators have not been independently validated. It must also be recognized that the measurement of lung mechanics from the proximal airway assumes that the lungs are being passively inflated—which is not the case with spontaneous breathing modes (SIMV or pressure support), and is not always the case with assisted ventilation (particularly if the flow is set too low).[14,15]

I believe that several conclusions about the delivery of aerosols in mechanically ventilated patients can be made from these studies:

- The delivery of aerosolized medications to intubated mechanically ventilated patients is not very efficient.
- The amount of aerosol delivered to the endotracheal tube can be increased by using appropriate diluent volumes and nebulizer flows, and by proper placement of the nebulizer in the ventilator circuit.
- The major barrier to efficient delivery of aerosolized medications to intubated patients is the endotracheal tube.
- Evaluation of bronchodilator response in intubated patients is difficult, but a physiologic effect from inhaled aerosols can be demonstrated.

Within the past 5 years, interest has been renewed in the use of metered dose inhalers to deliver aerosolized bronchodilators to ambulatory patients.[16–22] A number of studies have shown that use of an MDI, with or without an auxiliary device such as an InspirEase or AeroChamber, is as effective as a SVN in many ambulatory patients. The use of MDI with intubated mechanically ventilated patients has also been met with interest, and this has been the topic of several abstracts presented in recent years at the RESPIRATORY CARE Open Forum,[22–27] the American Thoracic Society,[28,29] American College of Chest Physicians,[30,31] and the Society for Critical Care Medicine.[32]

Although interest in the use of MDI with intubated patients in the critical care unit has only recently been aroused, it has existed for many years in anesthesia.[33–38] In 1977, Sprague described the use of an isoetharine MDI (Bronkometer) in 16 patients who developed bronchospasm during general anesthesia.[33] He reported a decrease in the severity of wheezing and in peak airway pressures as the result of Bronkometer use. Some more recent reports in the anesthesia literature[35–38] on the use of MDI with intubated patients, relate to techniques for inserting the MDI at the airway.

At the 1987 RESPIRATORY CARE Open Forum, Crogan and Bishop[22,39] reported the results of a bench study that evaluated the delivery efficiency of MDI-generated metaproterenol through an endotracheal tube. They found that the amount of aerosol delivered varied from 3.0 ± 1.9% for a 6-mm endotracheal tube to 6.5 ± 4.4% for a 9-mm tube. Significantly more aerosol traversed the endotracheal tube when the MDI was activated into a continuous flow of gas (analogous to actuation of the MDI near the beginning of the inspiratory phase of the ventilator), rather than actuation of the MDI before gas flow was initiated (analogous to actuation before the initiation of the inspiratory phase of the ventilator).

Fernandez et al[40] recently compared the use of ipratropium bromide by MDI, salbutamol by MDI, and aminophylline I.V., in patients with chronic obstructive pulmonary disease who were being mechanically ventilated. They found that these three drugs were equally effective in producing bronchodilatation, and concluded that MDI administration during mechanical ventilation is as effective as I.V. aminophylline. It is of interest to note that they used a self-inflating resuscitator bag and delivered the drug during a slow, deep breath (5-6 s, 1.5 L), followed by a 10-second breathhold. In order to accomplish this maneuver, they sedated and paralyzed the patients during the treatment.

At the 1988 RESPIRATORY CARE Open Forum, my co-workers and I reported results of a bench study[23] similar to that of Crogan and Bishop. In that study, we found 3.3 ± 2.1% of the MDI aerosol traversed the endotracheal tube, 7.4 ± 3.2% impacted in the MDI adapter, and 89.2 ± 3.8% impacted in the endotracheal tube. Impaction of aerosol in the

Fig. 1. Illustration of aerosol impaction in an endotracheal tube. Note evidence of decrease in density of aerosol down the length of the tube (from laboratory work done at York Hospital, York PA).

proximal endotracheal tube was visually apparent (Fig. 1). In that study, flow and tidal volume had no effect on the amount of aerosol traversing the endotracheal tube. Endotracheal tube size also had no effect, but tubes of only two sizes (7 mm and 9 mm) were used.

From these two bench studies, it is apparent that the endotracheal tube serves as a formidable barrier to aerosol from an MDI. However, deposition from an MDI with an endotracheal tube in place appears to be no worse than deposition from a SVN with an endotracheal tube in place. For example, MacIntyre et al[1] found that only $2.9 \pm 0.7\%$ of aerosol from a SVN traversed the endotracheal tube. With nonintubated subjects, only about 10% of MDI aerosol is deposited in the lungs, with the majority of MDI aerosol impacting in the oropharynx in these subjects.[41] Thus, one might conclude that MDI aerosol deposition in intubated patients is no worse than SVN aerosol deposition in intubated patients. Further, one might expect the fraction of the MDI aerosol dose delivered to the lungs in intubated patients to approach that delivered to the lungs of nonintubated patients if the dose (number of actuations) is doubled or tripled.

In the patient who is not intubated, aerosol from the MDI that impacts on the oropharynx is swallowed, may be systemically absorbed, and may produce bronchodilatation and systemic side effects. One can only speculate as to the fate of the aerosol that impacts on the endotracheal tube in intubated patients. Although much of it may be coughed or suctioned out of the tube, some of it may eventually enter the respiratory tract. This may be a cause for concern about side effects, although conventional wisdom holds that very little of the medication that enters the lungs is systemically absorbed (that's why we administer it by inhalation), and that current-generation bronchodilators are very safe—even at high doses. However, clear evidence for this 'wisdom' is lacking, and more study is needed. The most important point is that some of the MDI aerosol that impacts on the endotracheal tube may eventually enter the lungs and produce bronchodilatation.

At the 1989 RESPIRATORY CARE Open Forum, Larson et al[25] reported results with a bench model to compare the volume of particles delivered to the end of the endotracheal tube when several different MDI adapters were used. They found that each of the adapters that they evaluated allowed less aerosol to be delivered through the endotracheal tube than that delivered from a standard actuator (ie, the one used with the inhaler by ambulatory patients). They also found differences between the types of actuators and recommended that the number of puffs delivered when an MDI is used with intubated patients be increased (50% increase with the Monaghan AeroChamber In-Line Spacer, 350% for the Instrumentation Industries MDI adapter, and 800% for the Intec adapter).

At the 1988 Open Forum, Gutierrez and Nelson[24] reported their evaluation of SVN vs MDI in 20 intubated patients. Changes in airway resistance after administration of metaproterenol were similar for SVN and MDI. They concluded that SVN and MDI were equally capable of producing bronchodilatation in intubated mechanically ventilated patients.

At the 1989 Open Forum, my co-workers and I[26] reported the use of MDI with intubated patients—

comparing the use of SVN to MDI in 16 intubated mechanically ventilated adult patients. We evaluated breath sounds, peak pressure, pause pressure, inspiratory resistance, expiratory resistance, and compliance before and after each treatment, and also before and after a control evaluation in which no aerosol was administered. With the exception of expiratory resistance, the changes in measured variables were similar for MDI, SVN, and control. A significant decrease in expiratory resistance occurred with SVN and MDI but not with control. The mean decrease in expiratory resistance was 18% with MDI and 12% with SVN. Breath sounds improved in 7 patients with MDI, 7 patients with SVN, and 2 patients with control. It is important to note that we used 4 puffs of MDI for this study, rather than the 2 puffs commonly used in ambulatory patients. The ventilator settings used were a tidal volume of 12-15 mL/kg, inspiratory time of 25%, and pause time of 5%.

Also at the 1989 Open Forum, Bakow et al[27] compared the use of SVN and MDI in 30 intubated patients. They concluded that SVN and MDI can be used with equal effectiveness in intubated mechanically ventilated patients.

An interesting paradox surfaces when the results of bronchodilator administration by SVN and MDI are compared. The dose used with SVN is approximately ten times the dose used with MDI, but the bronchodilatation produced is similar for each method of administration (at least in ambulatory patients). For example, a 0.3 mL dose of metaproterenol (5% solution) is equivalent to 15 mg, whereas 2 puffs of metaproterenol (0.65 mg each) is equivalent to 1.3 mg. The dose from the SVN may need to be greater to compensate for the volume of drug retained in the nebulizer and the amount of drug lost during the patient's expiratory phase (if the nebulizer runs continuously, which is usually the case).

Fuller et al[42] compared the delivery of labeled fenoterol by MDI and by SVN in mechanically ventilated patients and found significantly greater deposition by the MDI (5.76% of the dose deposited in the lung) than by the SVN (1.22% of the dose deposited in the lung). For MDI delivery, they used an aerosol holding chamber, 15 cm from the endotracheal tube on the inspiratory limb. The MDI was actuated near the end of exhalation, without using an inspiratory pause or any other changes in ventilator settings. Four puffs of the MDI were used at 5-minute intervals.

From these bench and patient studies, one can conclude:

- A measurable amount of aerosol is delivered through the endotracheal tube when a MDI is used.
- Less MDI aerosol is deposited in the lungs through an endotracheal tube than is deposited in the lungs of a nonintubated patient.
- The response of an intubated patient to bronchodilator delivered by MDI is similar to the response to bronchodilator delivered by SVN.

Potential benefits of the use of MDI instead of SVN in intubated patients are:

- It takes only about half the time (or less) to deliver MDI compared to SVN—decreasing the cost of therapy and freeing the respiratory care practitioner to perform other duties.
- The continuous flow from the SVN can damage the expiratory flow transducer of the Servo 900C ventilator—a problem avoided by use of a MDI.
- As reported by Ritz et al at the 1989 Open Forum,[43] and subsequently in a full paper,[44] the continuous flow from a SVN can result in difficulty for some patients in triggering breaths during pressure support ventilation—a problem eliminated by the use of an MDI.
- Craven[45] has shown that the SVN can be the source of airway contamination in intubated patients—a source of infection eliminated by use of MDI. Further, use of a MDI may allow the patient to be treated without opening the ventilator circuit—avoiding possible aerosolization of organisms from the patient into the environment.

I believe that MDI can be safely and effectively substituted for SVN in mechanically ventilated intubated adult patients. At York Hospital, all inhaled bronchodilators for intubated mechanically ventilated patients are now administered by MDI.

Although I believe that evidence supports the use of MDI in place of SVN in intubated adult patients, several questions about this therapy remain unanswered:

- What are the best ventilator settings to use when delivering MDI therapy to intubated patients?

- In ambulatory patients, the MDI aerosol is delivered with the patient taking a slow, deep breath, followed by a breath-hold (for as long as 10 s). Is this desirable—or even possible—in intubated patients?
- What is the appropriate number of puffs from the MDI for intubated patients?
- How should the appropriate dose be determined?
- Should there be a standard dose or is the dose patient-dependent?
- Is systemic absorption and overdose a real concern?
- Are some commercially available MDI adapters clinically superior to others?
- How does one best evaluate patient response to MDI therapy, at the bedside?
- Can patient response be measured using the lung mechanics microprocessors available on some ventilators?
- Where in the ventilator circuit should the MDI be placed?
- What are the indications for aerosolized bronchodilator administration in mechanically ventilated patients?
- In patients with lung disease, is the aerosol delivered to the diseased areas of the lungs (which may need it most) or is the aerosol preferentially delivered to more normal areas?

I believe that these are questions that can (and should) be studied by respiratory care practitioners, physicians, and others interested in clinical aerosol therapy. Perhaps these questions will be answered in future years at the RESPIRATORY CARE Open Forum.

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Imposed Work of Breathing during Synchronized Intermittent Mandatory Ventilation Provided by Five Home Care Ventilators

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The imposed inspiratory work of breathing with mechanical ventilators designed for critical care has been extensively studied. However, no data concerning imposed inspiratory work of breathing using home care ventilators has been published. METHODS AND MATERIALS: We measured the imposed inspiratory work of breathing and the maximum negative pressure deflection during spontaneous inspiration with five home care ventilator systems, using a lung model. The Aequitron LP-6, Puritan-Bennett 2800, Lifecare PLV-100, Bear Medical 33, and Medimax ARF-1500E home care ventilators with standard circuits were compared to the Lifecare PLV-100. The PLV-100 was equipped with a one-way H-valve between the ventilator and humidifier that allowed spontaneous breathing from atmosphere or from a reservoir setup, and avoiding the ventilator's internal circuitry. Data were collected from six simulated spontaneous breaths for each system, at various peak inspiratory flowrates with three humidification systems (bubble-through, pass-over, and heat-and-moisture exchanger). RESULTS: Imposed inspiratory work of breathing and maximum negative pressure deflection increased as peak flow increased and with increased resistance to inspiration (bubble-through > exchanger > pass-over). The imposed inspiratory work of breathing and maximum pressure deflection were significantly greater with all systems when compared to the H-valve modifications with either the pass-over or the heat-and-moisture exchanger humidifiers. CONCLUSION: As a result of the increased inspiratory work of breathing observed in the five home care ventilators during simulated spontaneous breathing, we caution against the use of these home care ventilators in the SIMV mode unless appropriate circuit modifications are made. (Respir Care 1990;35:405-414.)

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Introduction

The number of patients who receive home mechanical ventilation increases yearly. It was estimated during the late 1980s that approximately 7,000 individuals in the United States, and additional thousands in Europe, were receiving ventilatory support in the home.[1] As a result, a number of new, primarily microprocessor-based home mechanical ventilators have been developed and marketed. These ventilators are more sophisticated than the previous generation of home mechanical ventilators. Many of them provide extensive alarm systems and digital readouts and incorporate a number of modes of ventilation. Each includes, at a minimum, the control, assist-control, and intermittent (IMV) or synchronized intermittent (SIMV) mandatory ventilation modes. In the ICU setting, IMV/SIMV is normally accomplished by the incorporation of a demand system into the ventilator's gas delivery apparatus or the addition of a continuous gas flow system.[2] When we examined the internal operation of a number of the newer mechanical ventilators for home use and consulted the operating manuals, we could identify no demand or continuous gas flow system.[3–7] Fig. 1 illustrates the basic gas delivery system utilized in most home care ventilators. During spontaneous breathing in the IMV/SIMV mode, gas can be drawn

Abbreviations Used in this Paper

ACCP — American College of Chest Physicians
COPD — Chronic obstructive lung disease
HME — Heat-and-moisture exchanger
ICU — Intensive care unit
IMV — Intermittent mandatory ventilation
PEEP — Positive end-expiratory pressure
P_{max} — Maximum negative-inspiratory-pressure deflection
SIMV — Synchronized intermittent mandatory ventilation
V_T — Tidal volume
WOB_I — Inspiratory work of breathing

A Guide to the Use of SI in This Paper*

The SI unit for pressure is the kilopascal (kPa).
(cm H_2O)(0.098 06) = kPa.

The SI unit for compliance is liters per kilopascal (L/kPa).
(L/cm H_2O)(10.20) = L/kPa.

The SI unit for length is the meter (m).
(in)(0.025 4) = m.

*For further information on SI (le Système International d'Unités), see Respir Care 1988;33:861-873 (October 1988) and Respir Care 1989;34:145 (February 1989—Correction).

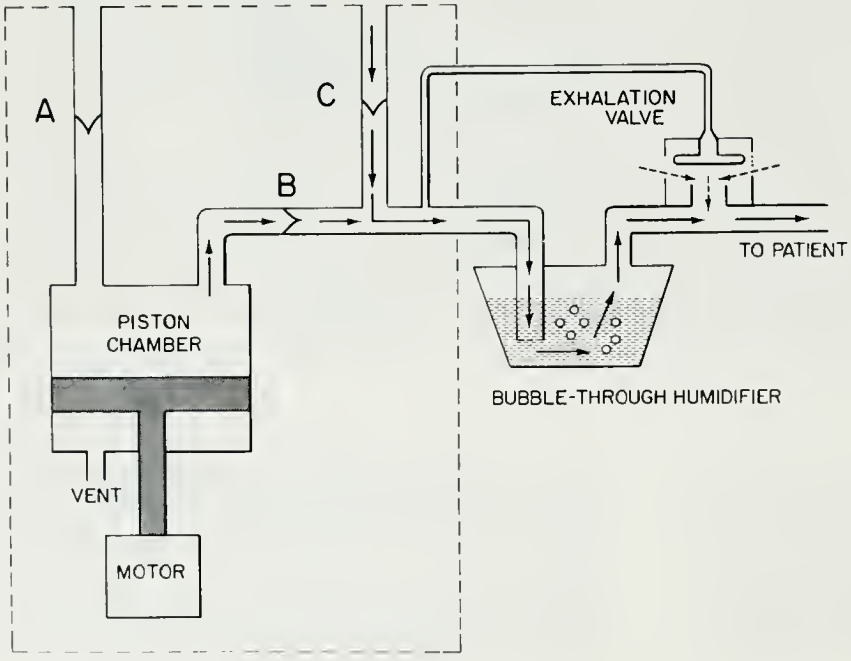

Fig. 1. Diagram showing gas flow during spontaneous inspiration through a typical home care ventilator. A. One-way check valve allows gas entry into the piston chamber during piston backstroke. B. One-way check valve prevents subatmospheric pressure from developing in the ventilator circuit during backstroke of piston. C. One-way check valve allows patient to inspire spontaneously during closure of B when piston backstroke is in progress. Some gas may enter system at the exhalation valve. Arrows depict gas flow during spontaneous inspiration.

into the inspiratory circuit from (1) the air-intake valve of the piston chamber via the piston chamber itself (however, gas flow from this route is not available, during the backstroke of the piston); (2) through an antisuffocation valve located within the machine between the piston and the exit port for gas flow from the ventilator; and (3) via the exhalation valve of the ventilator circuit.

The inspiratory work of breathing (WOB_I) imposed by ICU ventilators during spontaneous breathing has been extensively studied and documented.[8-10] However, no data have been published on the work of breathing imposed by home mechanical ventilators during the spontaneous breathing phase of IMV/SIMV. Using a lung model, we evaluated the WOB_I imposed by the Aequitron LP-6, Puritan-Bennett 2800, Bear 33, Lifecare PLV-100, and the Medimax ARF-1500E, during the spontaneous phase of IMV/SIMV.*

Methods and Materials

We evaluated the WOB_I and the maximum negative-inspiratory-pressure deflection (P_{max}) during simulated spontaneous breathing in IMV/SIMV modes in the Aequitron LP-6, Puritan-Bennett 2800, Bear 33, Lifecare, PLV-100, and Medimax ARF-1500E. We tested these ventilators during spontaneous ventilation simulated by a lung model (Fig. 2) previously described by Op't Holt et al[11] and Katz et al.[9] The model consisted of a two-chambered Michigan Instruments training test lung. One chamber of the test lung was attached to and powered by an Emerson ventilator (driving chamber); the other chamber (experimental chamber) was attached to the ventilator being studied. The two chambers were physically connected only by a small metal insert that allowed the driving chamber to lift the experimental chamber. Thus, the establishment of positive pressure in the driving chamber created subatmospheric pressure in the experimental chamber, simulating a spontaneous breath. The compliance of both chambers was set at 0.045 L/cm H_2O [0.46 L/kPa]. A standard-length 8-mm internal-diameter oral endotracheal tube was used to connect each ventilator to its respective chamber, and sufficient PEEP was applied to the driving chamber to prevent chamber separation at end-expiration. The circuit of the experimental ventilator was configured with a pressure tap and Hans Rudolph 3700 pneumotachograph in series between the distal end of the ventilator circuit and the proximal end of the endotracheal tube. The pressure tap led to a Validyne MP45-32-871 $\pm$ 100 cm H_2O differential pressure transducer. Pressure and flow signals were measured and integrated by a DEC LSI 11/23 computer with an ADAC data-acquisition module, and displayed as

*Suppliers are identified in the Product Sources section at the end of the text.

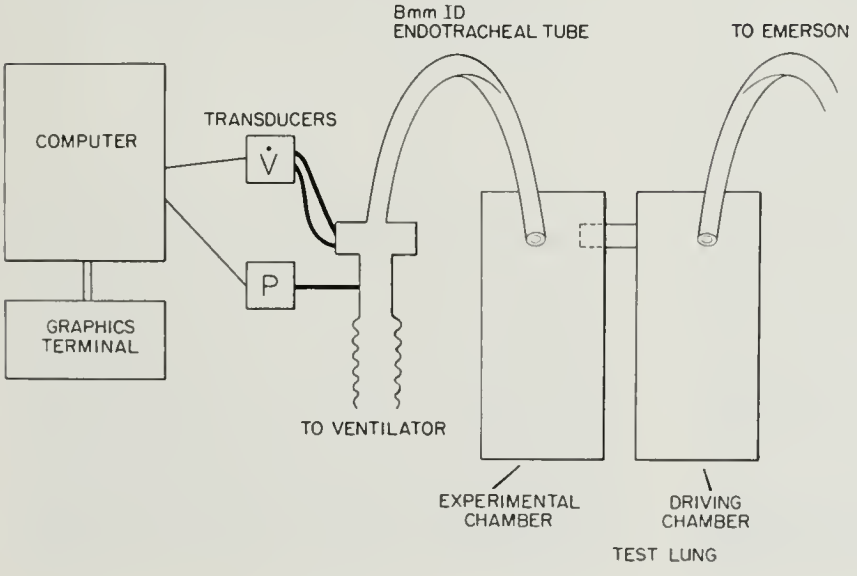

Fig. 2. Diagram of experimental setup showing lung model used to simulate spontaneous breathing, positions of pressure and flow transducers, and computer for data collection and analysis.

Table 1. Settings for Experimental Chamber of Test Lung prior to Ventilator Attachment

Inspiratory Peak Flow (L/min)	V_T (mL)	Inspiratory Time(s)
20	350	1.6
30	375	1.2
40	500	1.2
50	525	1.0
60	550	0.9

pressure-volume loops on a Tektronix 40006-1 graphics terminal. The raw data and the pressure-volume loops were analyzed by computer. The WOB_I and P_{max} were determined on the basis of measurements made at the junction of the ventilator circuit and the endotracheal tube. The Emerson ventilator that powered the driving chamber was set to ensure the series of peak flows, tidal volumes, and inspiratory times listed in Table 1, prior to the attachment of the ventilator to be tested.

All ventilators were evaluated at each combination of listed settings (Table 1) with humidification provided by pass-over (Puritan-Bennett Cascade 1 without tower), bubble-through (Puritan-Bennett Cascade 1 with tower), and heat-and-moisture exchanger (HME) (Siemens SH 150) humidifiers. In addition, the PLV-100 was configured in two ways with a one-way valve (H-valve) between the ventilator and the system humidifier (Fig. 3): (1) valve open to atmosphere and (2) valve with a 3-L reservoir bag attached to a 28% oxygen air-entrainment valve, powered by an oxygen flow of 4 L/min. The gas exiting the system was analyzed and varied about 28% by ± 2%. Each of these one-way (H) valve setups was evaluated at all settings (Table 1) of the experimental chamber and with all three humidification systems. Each experimental ventilator was set up according to manufacturer's instructions.[3-7] All were set in the SIMV mode (the Medimax ARF-1500E only provides IMV by setting the ventilator in the control mode) at the minimum positive-pressure rate available. Trigger sensitivity was set at maximum level (–1 cm H_2O [–0.1 kPa]) in all units, except the ARF-1500E (in the IMV mode, the mode selector is set in the control mode and the ventilator does not respond to inspiratory effort).

Data were collected for six simulated spontaneous inspirations at each experimental condition. Mean and

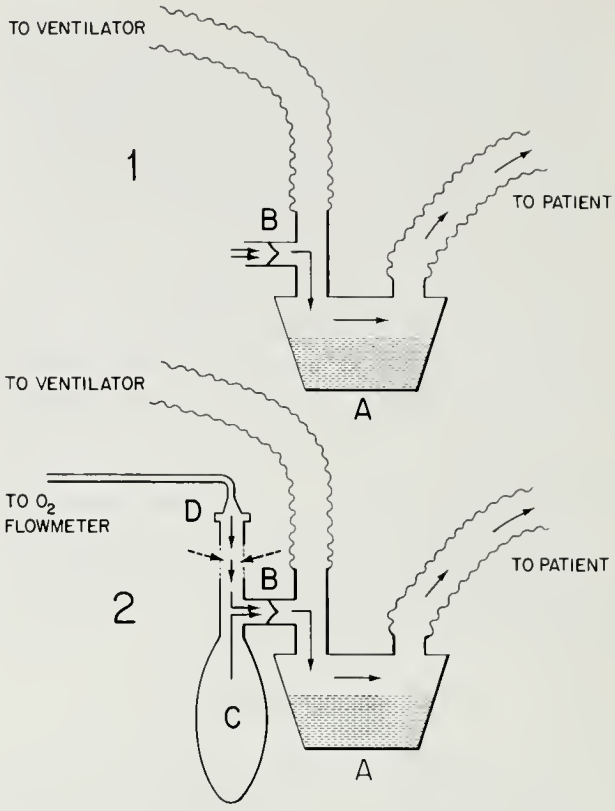

Fig. 3. Diagram of one-way H-valve systems: 1—valve open to atmosphere; 2—valve with 3-L reservoir bag attached to 28% oxygen air-entrainment device, powered by 4 L/min oxygen. A—pass-over humidifier. B—one-way valve. C—reservoir. D—28% oxygen air-entrainment valve. Arrows depict gas flow during spontaneous inspiration.

standard deviations were calculated for each set of data. Significance ($p < 0.05$) was evaluated by one-way ANOVA; post-hoc analysis was performed by Newman-Keuls test.

Results

The data obtained during all evaluations are summarized in Tables 2-5 and Figures 4-7. Of all the systems, the H-valve continuous-flow system with the pass-over humidifier imposed the least WOB_I and required the least negative airway pressure change ($p < 0.05$, Fig. 7, Table 5). However, no statistical differences in the variables measured were found with the pass-over humidifier or the HME and either H-valve setup. The bubble-through Cascade system always imposed a greater WOB_I and required a greater inspiratory effort than did the HME or pass-

Table 2. Inspiratory Work of Breathing (WOB_I) Imposed and Maximum Negative Pressure (P_{max}) Required for Spontaneous Breaths with Pass-Over Humidifier

	Ventilator									
	Bear 33		P-B 2800		LP-6		1500-E		PLV-100	
Flow L/min	WOB_I* J/L	P_{max} cm H_2O†	WOB_I J/L	P_{max} cm H_2O	WOB_I J/L	P_{max} cm H_2O	WOB_I J/L	P_{max} cm H_2O	WOB_I J/L	P_{max} cm H_2O
20	0.077 (0.005)	-1.75 (0.266)	0.180 (0.000)	-2.28 (0.126)	0.183 (0.005)	-2.45 (0.214)	0.141 (0.000)	-3.10 (0.197)	0.148 (0.008)	-3.52 (0.261)
30	0.133 (0.019)	-2.75 (0.163)	0.179 (0.001)	-3.05 (0.151)	0.169 (0.008)	-3.22 (0.103)	0.215 (0.005)	-4.98 (0.371)	0.202 (0.009)	-4.45 (0.321)
40	0.165 (0.009)	-3.72 (0.201)	0.210 (0.003)	-3.76 (0.243)	0.266 (0.005)	-5.38 (0.133)	0.278 (0.004)	-6.36 (0.214)	0.316 (0.010)	-6.24 (0.335)
50	0.180 (0.008)	-5.27 (0.234)	0.193 (0.005)	-4.12 (0.331)	0.277 (0.005)	-6.33 (0.271)	0.267 (0.005)	-6.95 (0.226)	0.393 (0.005)	-9.95 (0.187)
60	0.220 (0.006)	-6.08 (0.879)	0.220 (0.002)	-5.80 (0.803)	0.290 (0.000)	-7.68 (0.232)	0.287 (0.005)	-8.83 (0.172)	0.450 (0.013)	-12.41 (0.138)

*All values in all columns differed significantly ($p < 0.05$) from the H-valve system with a pass-over humidifier or HME. All values are mean (SD).
†Refer to box for SI conversion factors.

Table 3. Inspiratory Work of Breathing (WOB_I) Imposed and Maximum Negative Pressure (P_{max}) Required for Spontaneous Breaths with Heat-and-Moisture-Exchanger

	Ventilator									
	Bear 33		P-B 2800		LP-6		1500-E		PLV-100	
Flow L/min	WOB_I* J/L	P_{max} cm H_2O†	WOB_I J/L	P_{max} cm H_2O	WOB_I J/L	P_{max} cm H_2O	WOB_I J/L	P_{max} cm H_2O	WOB_I J/L	P_{max} cm H_2O
20	0.137 (0.005)	-2.22 (0.186)	0.195 (0.005)	-3.13 (0.308)	0.184 (0.008)	-2.62 (0.243)	0.157 (0.003)	-3.50 (0.175)	0.180 (0.018)	-4.00 (0.295)
30	0.145 (0.018)	-2.81 (0.618)	0.190 (0.002)	-3.85 (0.152)	0.178 (0.004)	-3.25 (0.176)	0.221 (0.008)	-5.05 (0.226)	0.243 (0.014)	-5.21 (0.310)
40	0.187 (0.005)	-5.83 (0.331)	0.220 (0.006)	-4.68 (0.241)	0.276 (0.005)	-5.82 (0.175)	0.290 (0.006)	-6.58 (0.264)	0.345 (0.018)	-6.37 (0.441)
50	0.218 (0.004)	-6.45 (0.459)	0.198 (0.004)	-4.98 (0.417)	0.280 (0.004)	-6.13 (0.293)	0.283 (0.005)	-7.78 (0.183)	0.435 (0.009)	-10.98 (0.271)
60	0.236 (0.005)	-6.15 (0.387)	0.228 (0.008)	-5.35 (0.337)	0.340 (0.006)	-9.65 (0.900)	0.324 (0.005)	-9.32 (0.155)	0.475 (0.005)	-12.97 (0.266)

*All values in all columns differed significantly ($p < 0.05$) from the H-valve system with a pass-over humidifier or HME. All values are mean (SD).
†Refer to box for SI conversion factors.

over humidifier regardless of ventilator or H-valve evaluated (Figs. 4-7). Performance was similar in all of the ventilators, although the WOB_I and P_{max} were least for the Bear 33 and greatest for the PLV-100, regardless of humidifying system or peak inspiratory flow. All units imposed greater WOB_I and required higher P_{max} as peak inspiratory flowrates increased.

Discussion

The most important finding of this study was the level of WOB_I and the magnitude of negative inspiratory pressure developed during simulated spontaneous ventilation in the IMV/SIMV modes of all the units studied when standard ventilator circuits

Table 4. Inspiratory Work of Breathing (WOB_I) Imposed and Maximum Negative Pressure (P_{max}) Required for Spontaneous Breaths with Bubble-Through Humidifier System

	Ventilator									
	Bear 33		P-B 2800		LP-6		1500-E		PLV-100	
Flow L/min	WOB_I* J/L	P_{max} cm H_2O†	WOB_I J/L	P_{max} cm H_2O	WOB_I J/L	P_{max} cm H_2O	WOB_I J/L	P_{max} cm H_2O	WOB_I J/L	P_{max} cm H_2O
20	0.346 (0.005)	−4.98 (0.248)	0.332 (0.004)	−4.70 (0.155)	0.346 (0.005)	−4.66 (0.226)	0.335 (0.010)	−6.97 (0.301)	0.364 (0.005)	−6.32 (0.141)
30	0.317 (0.015)	−4.83 (0.869)	0.348 (0.008)	−5.56 (0.242)	0.283 (0.027)	−5.88 (0.204)	0.407 (0.012)	−8.52 (0.229)	0.406 (0.018)	−7.93 (0.367)
40	0.340 (0.022)	−6.40 (0.237)	0.385 (0.005)	−6.82 (0.354)	0.383 (0.008)	−8.38 (0.179)	0.437 (0.010)	−9.37 (0.197)	0.465 (0.010)	−9.18 (0.601)
50	0.314 (0.004)	−7.82 (0.416)	0.335 (0.008)	−7.95 (0.266)	0.375 (0.009)	−9.55 (0.423)	0.417 (0.012)	−10.97 (0.423)	0.573 (0.010)	−13.95 (0.217)
60	0.352 (0.005)	−8.58 (0.306)	0.364 (0.005)	−7.94 (0.327)	0.497 (0.008)	−12.86 (0.258)	0.442 (0.004)	−12.37 (0.255)	0.616 (0.015)	−16.53 (0.240)

*All values in all columns differed significantly ($p < 0.05$) from the H-valve system with a pass-over humidifier or HME. All values are mean (SD).
†Refer to box for SI conversion factors.

Table 5. Inspiratory Work of Breathing (WOB_I) Imposed and Maximum Negative Pressure (P_{max}) Required during Spontaneous Breaths for H-Valve Systems with Continuous Flow or Open to Atmosphere

	Humidifying Device											
	Pass-over				HME				Bubble-Through			
	Continuous Flow		Atmosphere		Continuous Flow		Atmosphere		Continuous Flow		Atmosphere	
Flow L/min	WOB_I J/L	P_{max} cm H_2O*	WOB_I J/L	P_{max} cm H_2O	WOB_I J/L	P_{max} cm H_2O	WOB_I J/L	P_{max} cm H_2O	WOB_I† J/L	P_{max}† cm H_2O	WOB_I† J/L	P_{max}† cm H_2O
20	0.009 (0.001)	−1.20 (0.089)	0.021 (0.004)	−1.32 (0.041)	0.019 (0.004)	−1.22 (0.041)	0.030 (0.000)	−1.2 (0.041)	0.231 (0.010)	−4.60 (0.210)	0.238 (0.005)	−4.90 (0.137)
30	0.024 (0.005)	−1.34 (0.052)	0.038 (0.004)	−1.45 (0.055)	0.030 (0.009)	−1.52 (0.041)	0.042 (0.036)	−1.6 (0.176)	0.243 (0.005)	−5.32 (0.041)	0.260 (0.009)	−4.87 (0.183)
40	0.035 (0.005)	−1.51 (0.041)	0.042 (0.006)	−1.83 (0.137)	0.051 (0.004)	−1.83 (0.052)	0.061 (0.008)	−2.2 (0.082)	0.260 (0.009)	−5.28 (0.052)	0.281 (0.018)	−5.73 (0.052)
50	0.046 (0.005)	−2.42 (0.052)	0.054 (0.004)	−2.80 (0.089)	0.058 (0.004)	−2.50 (0.089)	0.069 (0.003)	−3.1 (0.141)	0.281 (0.008)	−5.74 (0.052)	0.278 (0.005)	−5.97 (0.052)
60	0.053 (0.005)	−2.61 (0.052)	0.068 (0.011)	−3.08 (0.129)	0.062 (0.005)	−2.81 (0.041)	0.083 (0.005)	−3.8 (0.390)	0.280 (0.009)	−6.21 (0.041)	0.298 (0.023)	−7.81 (0.082)

*Refer to box for SI conversion factors.
†All values in column differed significantly ($p < 0.05$) from the H-valve system with a pass-over humidifier or HME. All values are mean (SD).

were used. WOB_I and P_{max} were elevated, regardless of humidifier employed. However, the pass-over humidifier and HME affected WOB_I and P_{max} the least (Tables 2-4 and Figs. 4-6). Of secondary importance was the finding that the WOB_I and P_{max} could be markedly reduced with the use of a one-way H-valve system inserted between the ventilator and a pass-over or HME humidifier (Table 5, Fig. 7). It is also important to note that this study was performed with a laboratory test lung under ideal conditions—properly functioning equipment with no water accumulation in the circuit. As a result, the

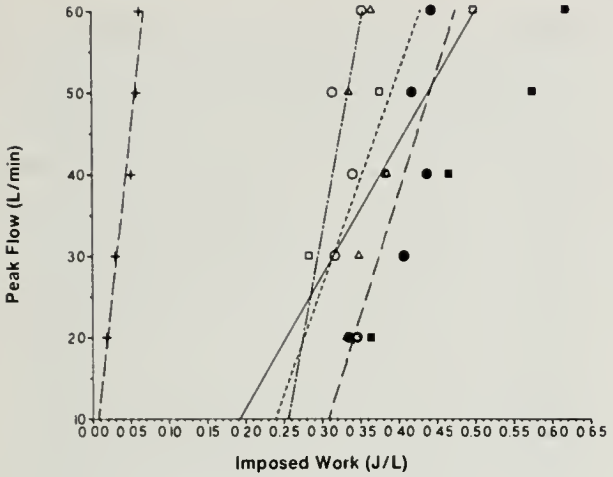

Fig. 4. Imposed work of breathing vs peak inspiratory flow for all ventilators evaluated with a normally functioning cascade humidifier (bubble-through) and the H-valve continuous-flow system with a Cascade humidifier modified as a pass-over humidifier. All points represent mean values, and lines are linear regression lines. Continuous-flow system with pass-over humidifier = + and —·—. Bear 33 = — - — and ○. Puritan-Bennett 2800 = - - - - and △. Aequitron LP-6 = —— and □. Medimax ARF-1500E = ——— and ●. Lifecare PLV-100 = ···· and ■.

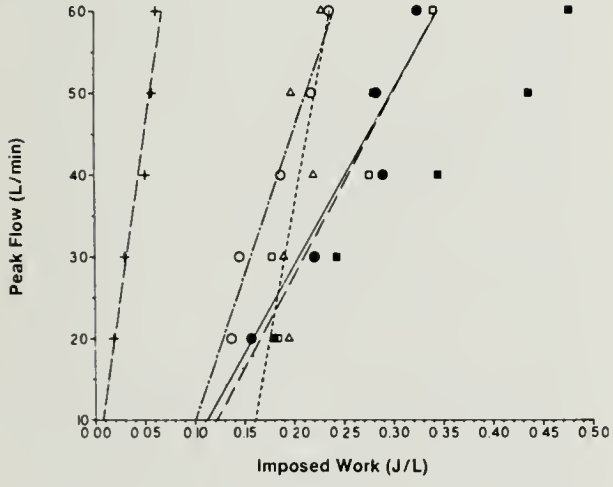

Fig. 5. Imposed work of breathing vs peak inspiratory flow for all ventilators evaluated with a heat-and-moisture exchanger and the H-valve continuous-flow system with a Cascade humidifier modified as a pass-over humidifier. All points represent mean values, and lines are linear regression lines. H-valve continuous-flow system with pass-over humidifier = + and —·—. Bear 33 = — - — and ○. Puritan-Bennett 2800 = - - - - and △. Aequitron LP-6 = —— and □. Medimax ARF-1500E = ——— and ●. Lifecare PLV-100 = ···· and ■.

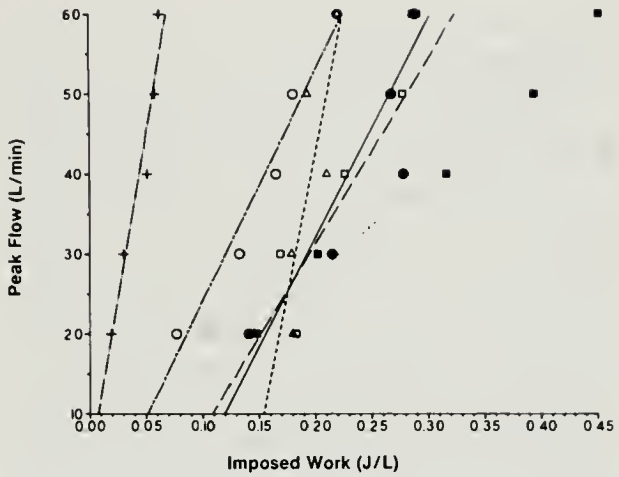

Fig. 6. Imposed work of breathing vs peak inspiratory flow for all ventilators evaluated with a Cascade humidifier modified as a pass-over humidifier system and the H-valve continuous-flow system with a Cascade humidifier modified as a pass-over humidifier. All points represent mean values, and lines are linear regression lines. H-valve continuous-flow system with pass-over humidifier = + and —·—. Bear 33 = — - — and ○. Puritan-Bennett 2800 = - - - - and △. Medimax ARF-1500E = ——— and ●. Lifecare PLV-100 = ···· and ■.

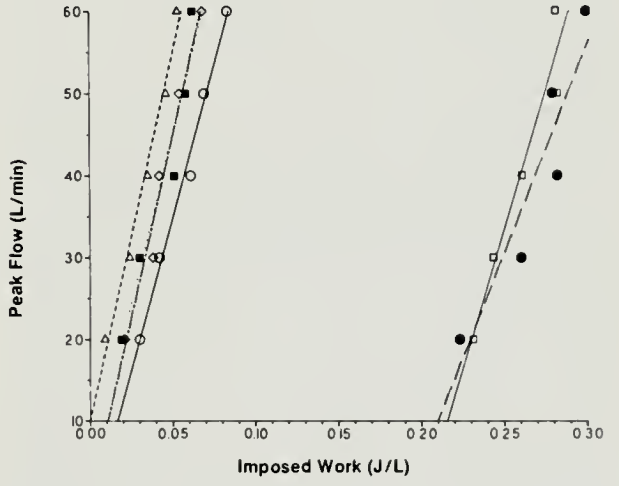

Fig. 7. Imposed work of breathing vs peak inspiratory flow for all six H-valve configurations. All points represent mean values, and lines are linear regression lines. H-valve with 28% oxygen air-entrainment system and pass-over humidifier = - - - and △. The H-valve with 28% oxygen air-entrainment system and the heat-and-moisture exchanger = · · · and ■. The H-valve open-to-atmosphere system and the pass-over humidifier = ◇ and —·—. The H-valve open-to-atmosphere system and the heat-and-moisture exchanger = ○ and ——. H-valve with 28% oxygen air-entrainment system and bubble-through humidifier = □ and ——. The H-valve open-to-atmosphere system and the bubble-through humidifier = ● and — — —.

data presented may underestimate the WOB_I and P_{max} encountered clinically.

All of these ventilators, when set up according to the manufacturers' recommendations, imposed WOB_I (0.332 to 0.616 J/L) essentially equal to the work associated with moving the lung in a normally healthy subject.[12] In addition, the P_{max} (−1.75 to −16.53 cm H_2O [−0.172 to −1.621 kPa]) generated for the most part exceeded the maximum recommended (−2 cm H_2O [−0.2 kPa]) airway pressure deflection[13,14] for spontaneous ventilation in the IMV/SIMV mode. Because patients requiring chronic ventilatory support may have ventilatory work loads two to three times greater than normal,[15,16] the additional imposed WOB_I during IMV/SIMV with these ventilators may be sufficient to invoke ventilatory muscle fatigue, especially at low mechanical breathing rates.[17,18]

Intensive care ventilators do impose inspiratory work during the spontaneous breathing phase of SIMV,[8–10] however, the levels imposed by the newer ICU ventilators are much lower than those imposed by the home care ventilators studied. A paper by Samodelov and Falke,[19] and an abstract by my co-workers and myself[20] have demonstrated the work loads imposed by ICU ventilators to be much less than those reported here. In fact, in our evaluation (using the same lung model) of four new ICU ventilators set at peak inspiratory flows of 40 L/min,[20] we observed WOB_I to be in 0.022 to 0.093 J/L range (about one tenth of that measured in the present study of home care ventilators) and P_{max} to be in the −1.25 to −3.10 cm H_2O [−0.123 to −0.304 kPa] range. The primary reason for these marked differences appears to be the difference between ICU and home care ventilators in the manner in which access to gas is gained during spontaneous ventilation. The accepted definitions for IMV/SIMV include access to either a continuous flow of gas delivered past the patient's airway sufficient to meet the patient's inspiratory demands or the inclusion of a demand gas delivery system.[2,21] None of the available home care ventilators tested met either of these criteria. All require patients to draw gas during spontaneous inspiration either from (1) the air-intake valve for the piston chamber via the piston chamber itself, (2) an antisuffocation valve located within the machine between the piston and the exit port for gas flow from the ventilator, or (3) the exhalation valve of the ventilator circuit (Fig. 1). If a bubble-through humidifier is used, gas must be drawn through a 2- to 3-in [0.05- to 0.08-m] column of water.

The Puritan-Bennett Cascade humidifier without its tower (ie, functioning as a pass-over humidifier) represents the function of each ventilator without additional impedance to spontaneous inspiration. Such a humidification system does not include valves, filters, or columns of water through which gas must be inhaled. In addition, the internal diameters of components of these systems through which gas normally passes are equal to or exceed the diameter of large-bore ventilator tubing. As a result, we presumed that the pass-over humidifier would not impose WOB_I regardless of the ventilatory condition evaluated. However, none of the ventilators were evaluated in the absence of a humidifier. In spite of the fact that the performance of the Siemens SH 150 HME was similar to that of the pass-over humidifier, the same cannot be said for the HME under all ventilatory conditions. As demonstrated by Ploysongsang et al,[22] impedance to gas flow in HMEs increases as peak flow increases and the HME becomes saturated with water vapor. We evaluated the performance of only one HME (Siemens SH 150) at low peak inspiratory flowrates (20-60 L/min) under dry conditions for short periods of time. The effect of Siemens SH 150 on WOB_I at higher flows or when saturated with water may vary greatly from our data. In addition, as demonstrated by Ploysongsang et al,[22] a large variance in resistance to gas flow exists among available HMEs.

If the IMV/SIMV modes on home care ventilators are used, the insertion of a one-way H-valve system distal to a pass-over humidifier or an HME can markedly reduce the WOB_I (0.009 to 0.083 J/L, Table 5) and the P_{max} (−1.20 to −3.8 cm H_2O, Table 5). These levels of WOB_I and P_{max} are consistent with those of ICU ventilator systems.[20] The addition of a continuous-flow reservoir system equipped with an air-entrainment device for control of oxygen concentration further improved the function of the H-valve but not significantly (Table 5).

Based on the results of our evaluation of home-care ventilator function, we do not recommend the use of the IMV/SIMV mode, particularly at low mechanical rates, unless the humidifier is of a pass-over or HME type and a one-way H-valve is incorporated into the system. Even with these modifications, the wisdom of employing the IMV/

SIMV mode in chronically ventilator-dependent subjects must be questioned.

The American College of Chest Physicians (ACCP)[23] has recommended that IMV/SIMV not be used during home mechanical ventilation because the use of these modes "may predispose the ventilatory muscles to fatigue, which worsens the existing chronic respiratory failure." Particularly in the adult population, the likelihood of weaning from chronic ventilatory support is small. We believe, as does the ACCP, that efforts should be directed at maximizing ventilatory muscle rest during periods of ventilatory support. This should help to enhance ventilatory muscle function during those periods of ventilator independence. The need for complete rest or at least minimal ventilatory muscle activity has been emphasized recently in the literature on noninvasive nocturnal ventilatory support. Recent reports by Kerby et al,[24] Carroll and Branthwaite,[25] and especially Leger et al[26] concerning nocturnal nasal positive-pressure ventilation have stressed the need for rest with the use of control or assist-control modes at backup rates that ensure controlled ventilation during sleep. These authors have all demonstrated improved ventilatory muscle capability and improved gas exchange, in series of both COPD and restrictive-lung-disease patients.

We believe that the fact that IMV/SIMV modes are available on today's home care ventilators points out the inability of clinicians and manufacturers to differentiate between the needs of patients in the ICU and in the home. Mechanical ventilators for home use appear to mimic ICU ventilators; but, perhaps in an attempt to be cost-effective, manufacturers have included poorly designed systems. Home care ventilators appear to have been developed with the ICU clinician—not the home care patient—in mind. With the likelihood of increased numbers of ventilator-assisted patients in the home in the 1990s, it seems both clinically desirable and cost-effective to develop a home ventilator system with the particular needs of the home care patient in mind.

Summary and Conclusion

We have evaluated the WOB_I and P_{max} during IMV/SIMV in five home care ventilators using three humidification systems, and the effect of modifying standard circuitry with one-way H-valve systems. Our findings are (1) Home care ventilators impose WOB_I and require large P_{max} during the spontaneous breathing phase of IMV/SIMV; (2) the WOB_I and P_{max} are affected by the humidifier used (bubble-through > HME > pass-over); (3) WOB_I and P_{max} increases as peak inspiratory flow increases; (4) the use of a one-way H-valve with a HME or pass-over humidifier reduces WOB_I by a factor of 10 and decreases P_{max} to acceptable levels. We conclude that these home care ventilators should not be used in the IMV/SIMV mode unless an H-valve with pass-over humidifier or HME is appropriately placed in the system; and we do not recommend the use of a bubble-through humidifier unless the control or assist-control mode of ventilation is used.

ACKNOWLEDGMENTS

We thank David Romagnoli MBA RRT and Nick Barker LPN for their assistance with the statistical analysis and Gertrude Shaw, Peggy Gately, and Michelle Duran for their assistance with the preparation of the manuscript.

PRODUCT SOURCES

Ventilators:
Aequitron LP-6, Aequitron Medical Inc, Minneapolis MN
PB 2800, Puritan-Bennett Corp, Overland Park KS
Lifecare PLV-100, Lifecare, Lafayette CO
Bear 33, Bear Medical Systems, Riverside CA
Medimax ARF-1500E, Medimax Inc, West Hempstead NY
Emerson postoperative 3-PV ventilator, JH Emerson Co, Cambridge MA

Lung model:
TTL, Michigan Instruments Inc, Grand Rapids MI

Pneumotoachograph:
Hans Rudolph 3700, Hans Ruduolph Inc, Kansas City MO

Transducer:
Validyne MP45-32-871 ± H_2O, Validyne Engineering Corp, Northridge CA

Computer and Enhancements:
DEC LSI 11/23 with ADAC data-acquisition module, Digital Equipment Co, Boston MA
Tektronix 40006-1 graphics terminal, Tektronix Inc, Beaverton OR

Humidifying devices:
Cascade I, Puritan-Bennett Corp, Overland Park KS
Siemens Servo 150 heat-and-moisture exchanger, Siemens Life Support Systems, Schaumburg IL

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An Evaluation of the Respironics BiPAP Bi-Level CPAP Device for Delivery of Assisted Ventilation

David A Strumpf MD, Carol C Carlisle BA RN, Richard P Millman MD, Kirk W Smith BA RRT, and Nicholas S Hill MD

We evaluated the efficacy of the Respironics bi-level positive airway pressure (BiPAP) device, a continuous positive airway pressure (CPAP) blower that cyclically superimposes a pressure boost (PB) upon the baseline CPAP, in functioning as a ventilatory assist device. METHODS: We tested the device with an artificial lung model with normal lung compliance and airway resistance, ventilatory rates of 12-20/min, and PB levels up to 15 cm H_2O [1.5 kPa]. We also tested it on four patients who had been receiving home nocturnal nasal positive-pressure ventilatory assistance via either standard portable ventilators or nasal CPAP alone. RESULTS: When used with the artificial lung model at the above-described settings, the BiPAP device functioned as a time-cycled, pressure-limited ventilator. Further increases in PB, lung compliance, or airway resistance—or reductions in inspiratory time—resulted in flow-limitation as well. Patients found the BiPAP device as comfortable as their previous devices, or more so. After long-term nocturnal use of the BiPAP device, patients' P_{aCO_2} was either stabilized or improved. CONCLUSIONS: We conclude that the Respironics BiPAP device offers an effective, simple, lightweight, and less expensive alternative to standard portable ventilators for patients with chronic respiratory failure who receive intermittent ventilatory support via the nasal route. (Respir Care 1990;35:415-422.)

Introduction

Recent studies have suggested that nocturnal intermittent positive-pressure ventilation (IPPV) delivered via a nasal mask is effective in reversing chronic hypoventilation in patients with neuromuscular disease and kyphoscoliosis.[1-4] The IPPV for these patients is usually delivered by a standard portable ventilator identical to those used by patients who require continuous ventilatory support. Such ventilators are typically equipped with sophisticated alarms and backup systems that may add unnecessarily to the expense and complexity of delivering nocturnal ventilation to patients who require intermittent ventilatory assistance but are not ventilator-dependent. We report our evaluation of the Respironics BiPAP device, a bi-level nasal continuous positive airway pressure (CPAP) device that provides a simple, relatively inexpensive, and effective way to deliver positive-pressure ventilatory assistance to such patients.

Description of Device

The Respironics bi-level positive airway pressure (BiPAP) device* is a standard nasal CPAP blower

Dr Strumpf is Research Fellow, Ms Carlisle is Research Assistant, and Drs Millman and Hill are Associate Professors of Medicine—Division of Pulmonary and Critical Care Medicine, Rhode Island Hospital and Brown University, Providence, Rhode Island. When this study was performed, Mr Smith was New England distributor for Respironics Inc. This study was supported by a grant from Respironics Inc, Monroeville, Pennsylvania. None of the authors has a financial interest in the BiPAP device, except that Mr Smith represented Respironics when the study was performed.

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*Suppliers are identified in the Product Sources section at the end of the text.

Abbreviations Used in This Paper

APRV	— Airway pressure release ventilation
auto-PEEP	— Intrinsic, or unintended, positive end-expiratory pressure
BiPAP	— Bi-level positive airway pressure
CPAP	— Continuous positive airway pressure
EPAP	— Expiratory positive airway pressure
I/E ratio	— Inspiratory-to-expiratory-time ratio
IMV	— Intermittent mandatory ventilation
IPAP	— Inspiratory positive airway pressure
IPPV	— Intermittent positive-pressure ventilation
f	— Breathing frequency
P_{aCO_2}	— Arterial carbon dioxide tension (pressure)
P_B	— Pressure boost (difference between IPAP and EPAP)
PEEP	— Positive end-expiratory pressure
PIP	— Peak inspiratory pressure
%IPAP	— Proportion of each ventilatory cycle spent at IPAP
$\dot{V}_E$	— minute ventilation
V_T	— Tidal volume

A Guide to the Use of SI in This Paper†

The SI unit for compliance is liters per kilopascal (L/kPa).
(L/cm H_2O)(10.20) = L/kPa.

The SI unit for pressure is the kilopascal (kPa).
(cm H_2O)(0.098 06) = kPa.
(torr)(0.133 3) = kPa.

The SI unit for resistance is kilopascals per liter per second (kPa · s · L^{-1}).
(cm H_2O · s · L^{-1})(0.098 06) = kPa · s · L^{-1}.

†For further information on SI (le Systemé International d'Unités), see Respir Care 1988;33:861-873 (October 1988) and Respir Care 1989;34:145 (February 1989—Correction).

modified with a solenoid system that allows timed cyclical delivery of positive airway pressure at two different levels (Fig. 1). The BiPAP device is attached to standard tubing and masks used for nasal CPAP. Recessed controls on the rear of the device allow selection of four variables: (1) expiratory positive airway pressure (EPAP), ranging from 2 to 20 cm H_2O [0.2-2.0 kPa]; (2) inspiratory positive airway pressure (IPAP), ranging from 2 to 25 cm H_2O [0.2-2.5 kPa]; (3) frequency of cycling between IPAP and EPAP, ranging from 6 to 30 cycles/min; and (4) the %IPAP, which is the proportion of each cycle spent at IPAP, ranging from 10 to 90%. The difference between IPAP and EPAP is called the pressure boost (PB). The device can generate a peak flow of 180 L/min.

The device measures 38 × 23 × 20 cm, has a mass of 6 kg, and has no backup battery system or alarms. The units we tested functioned exclusively in the time-cycled mode, in which the frequency and %IPAP are fixed but the patient can take additional breaths between those supplied by the device, in a fashion analogous to the intermittent mandatory ventilation (IMV) mode on a standard ventilator. A newer version, the BiPAP S/T, which is now available, offers the time-cycled mode that our test units provided, but it also offers two spontaneous modes. In the S ("Spontaneous") mode, IPAP and EPAP are set by a caregiver, and the patient triggers inspiration and expiration in a manner similar to that used in pressure-support ventilation.[5] The T mode ("Time-backup") is like the S mode except that machine-generated breaths are delivered when the patient's respiratory rate falls below a set level.

Evaluation Methods

Artificial Lung Studies

We first studied the BiPAP device with the aid of a two-lung artificial lung model, the Vent-Aid TTL, which allowed for variation of lung compliance and airway resistance. Compliance was adjusted by

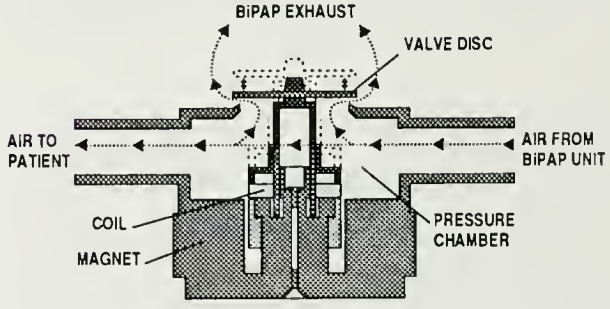

Fig. 1. Schematic diagram of BiPAP device's pressure-control mechanism. The coil and magnet cyclically apply a force to the valve disk that is equal to the IPAP and EPAP settings. Excess pressure is vented to the room, allowing for maintenance of stable pressures despite changes in flowrate.

changing the position of a precalibrated alloy spring, and resistance was varied by inserting metal cylinders of different internal diameters into the inflow tubing. 'Intrapulmonary pressure' was measured by a pressure transducer connected to an outlet port on the test lung. Airflow entering the lung was measured with a Fleisch pneumotachometer. Pressure and flow signals were recorded simultaneously on a two-channel recorder. Tidal volume (V_T) was measured with an Ohmeda volume meter connected to the inflow tubing.

We used the artificial lung to test the function of each control on the BiPAP device. First we tested the effect on delivered V_T when PB was increased, frequency and %IPAP were held constant, and lung compliance and airway resistance were 'normal.' Lung compliance was set at 0.10 L/cm H_2O [1.0 L/kPa], and airway resistance was set at 4.6 cm $H_2O \cdot s \cdot L^{-1}$ [0.45 kPa $\cdot$ s $\cdot$ L^{-1}] at a flowrate of 30 L/min, a value similar to that reported in healthy, conscious subjects in a tank respirator,[6] and in anesthetized, intubated patients receiving positive-pressure mechanical ventilation.[7] We then assessed the effects on delivered V_T when EPAP, %IPAP, and frequency were varied, still with normal compliance and resistance.

Having established the effects on delivered volume in a test lung with normal compliance and resistance, we then tested the BiPAP device against altered compliance and resistance, in order to mimic a number of pathologic conditions. Lung compliance was varied from 0.02 to 0.20 L/cm H_2O [0.2 to 2.0 L/kPa], and resistance was increased by lowering the internal diameter of the inflow tubing to each lung from 5.6 to 3.9 mm—more than a four-fold increase in pulmonary resistance.

Patient Studies

Four patients currently receiving nocturnal positive-pressure ventilatory assistance via a nose mask agreed to try the BiPAP device. They were drawn from the outpatient chest clinic at Rhode Island Hospital and gave informed consent prior to the BiPAP trial.

Patient 1 was a 57-year-old woman with both chronic bronchitis and severe restrictive lung disease (vital capacity, 0.40 L), the latter caused by osteogenesis imperfecta and kyphoscoliosis. She had been receiving nocturnal nasal ventilation via a Companion 2800 Home Ventilator for 6 months. After she had used the ventilator for 8 hours/night at a rate of 22 breaths/min and a V_T of 440 mL, her daytime P_{aCO_2} had fallen from a pre-ventilation level of 80 torr [11 kPa] to 51 torr [6.8 kPa] within 3 months.

Patient 2 was a 41-year-old woman with restrictive lung disease (vital capacity, 1.14 L) and chronic hypoventilation caused by muscular dystrophy. She had been using an LP-6 Home Ventilator for 5 months, 8 to 9 hours/night, at a rate of 15 breaths/min and a V_T of 500 mL. After 3 months of this, her daytime P_{aCO_2} had fallen from 55 to 48 torr [7.3 to 6.4 kPa].

Patient 3 was a 50-year-old man with restrictive lung disease (vital capacity, 1.35 L) and central respiratory depression due to post-poliomyelitis syndrome. After earlier using a cuirass ventilator for 4 years, he had most recently used nasal ventilation with a Thompson-Bantam Ventilator for 14 months. On settings of 15 breaths/min and an inspiratory pressure of 15 cm H_2O [1.5 kPa], his P_{aCO_2} had declined from 76 to 44 torr [10 to 5.9 kPa].

Patient 4 was a 31-year-old man with restrictive lung disease (vital capacity, 2.25 L) and obstructive sleep apnea (apnea-hypopnea index, 45/h) due to limb-girdle muscular dystrophy. His symptoms had failed to resolve on nasal CPAP alone despite suppression of apneic events with 12.5 cm H_2O [1.23 kPa] pressure, and his P_{aCO_2} remained at 56 torr [7.5 kPa].

The BiPAP device was taken to the patients' homes, where breathing rate, exhaled V_T, and minute volume were measured by an Ohmeda volume meter placed between the nasal mask and the nonrebreathing valve. Measurements were made during spontaneous and assisted breathing, with the patients using their standard ventilators (Patients 1-3) or CPAP device (Patient 4). Patients 1-3 were then connected to the BiPAP device, with frequency the same as with their standard ventilator and with EPAP set at 2 cm H_2O [0.2 kPa]. The %IPAP was set at 40%, which was virtually identical to that of their standard ventilators in all cases. The PB was then gradually increased until measured exhaled volumes were roughly equivalent to those generated by the standard ventilators. In Patient 4, the EPAP was set at 8 cm

H_2O [0.8 kPa] in order to overcome obstructive apneas, and the frequency and PB were adjusted to augment his spontaneous daytime minute ventilation by 20-30%.

Three patients requested long-term trials of the BiPAP device—Patient 2 because the low-pressure alarm on her standard ventilator was sounding intermittently and interfering with her sleep, and Patient 3 because the lesser weight of the BiPAP device facilitated transportation. Patient 4 was started on the BiPAP device after failing to improve on CPAP alone.

The BiPAP devices were set to provide minute volumes comparable to those delivered by the patients' previous ventilatory systems. Arterial blood samples for blood gases and pH analysis were collected within an hour after cessation of nocturnal ventilator use, before starting use of the BiPAP device, and periodically thereafter. The samples were transported on ice to the hospital laboratory for analysis.

Results of Test-Lung Evaluation

Effects of EPAP and Pressure Boost

Changing the EPAP setting altered the end-expiratory volume of the artificial lung, but did not influence the V_T as long as PB remained the same (data not shown). Figure 2 shows the V_T response of the test lung as PB was increased from zero to 20 cm H_2O [zero to 2.0 kPa], with frequency being

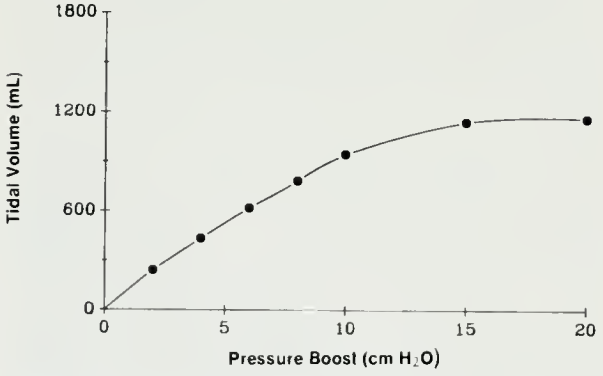

Fig. 2. Tidal volume of the lung model is plotted as pressure boost is increased from 0 to 20 cm H_2O. Frequency is 16/min, EPAP 5 cm H_2O, %IPAP 40%, lung compliance 0.10 L/cm H_2O, and airway resistance 4.6 cm $H_2O \cdot s \cdot L^{-1}$. [Refer to box for SI conversion factors.]

16/min, %IPAP 40%, EPAP 5 cm H_2O [0.5 kPa], resistance 4.6 cm $H_2O \cdot s \cdot L^{-1}$ [0.45 kPa $\cdot$ s $\cdot$ L^{-1}], and compliance 0.10 L/cm H_2O [1.0 L/kPa]. Up to a PB of 15 cm H_2O [1.5 kPa], V_T rises in proportion to PB. Above that amount of PB, flowrate becomes a limiting factor and V_T increases no further.

Effects of %IPAP and Rate Controls

Figure 3 shows the effect on V_T when %IPAP was increased at various PB levels, with other settings

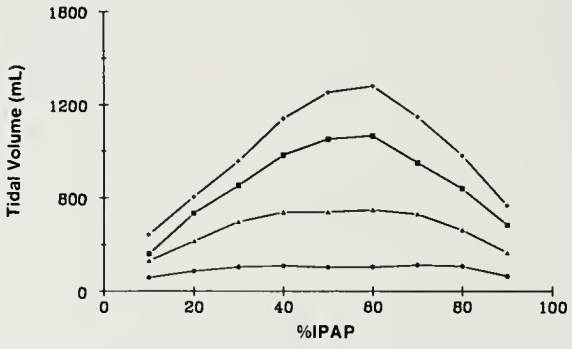

Fig. 3. Tidal volume of the lung model is plotted as %IPAP and PB are varied on the BiPAP device. —◆— PB 15 cm H_2O, —■— = PB 10 cm H_2O, —▲— = PB 5 cm H_2O, —•— = 2 cm H_2O. Frequency is 16/min, EPAP 5 cm H_2O , lung compliance 0.10 L/cm H_2O, and airway resistance 4.6 cm $H_2O \cdot s \cdot L^{-1}$. [Refer to box for SI conversion factors.]

kept constant, as indicated. A low %IPAP limits delivery of V_T, particularly at higher PB levels, because the brief duration of airflow does not allow the device to reach the preset pressure level. As %IPAP is increased above 60%, delivered V_T falls once again because the brief duration of expiration precludes complete emptying of the artificial lung. This results in the development of intrinsic positive end-expiratory pressure (intrinsic PEEP, or auto-PEEP) and higher end-expiratory volumes.

Figure 4 demonstrates that delivered V_T depends on both absolute inspiratory time during a single breath and the level of PB. As inspiratory time increases, smaller tidal volumes are actually delivered. Therefore, an increase in rate without a change in %IPAP will not necessarily increase minute volume—because the shortened absolute inspiratory time with each breath may diminish V_T.

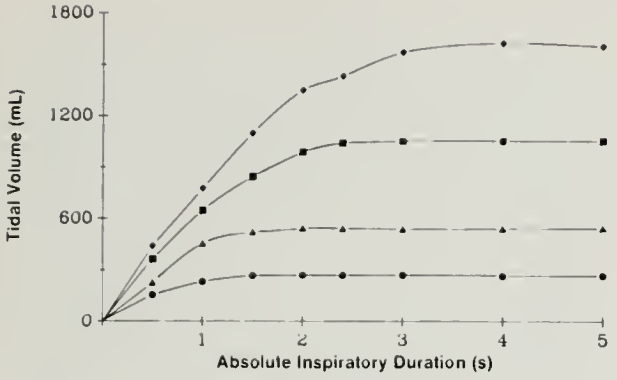

Fig. 4. Tidal volumes of the lung model at increasing durations of inspiratory time at various PB levels. —◆— = PB 15 cm H_2O, —■— = PB 10 cm H_2O, —▲— = PB 5 cm H_2O, —•— = 2 cm H_2O. EPAP is 5 cm H_2O, lung compliance 0.10 L/cm H_2O, and airway resistance 4.6 cm $H_2O \cdot s \cdot L^{-1}$. [Refer to box for SI conversion factors.]

Effects of Alteration in Lung Compliance

Figure 5 shows the effect on inspiratory airflow when lung compliance is decreased in a lung ventilated at 20 breaths/min, with EPAP of 5 cm H_2O [0.5 kPa], PB of 10 cm H_2O [0.98 kPa], and %IPAP set at 40%. In Panel A, representing a noncompliant lung, intrapulmonary pressure rapidly reaches the preset pressure limit, but flowrate peaks early and rapidly diminishes, and the V_T remains quite small. In Panel B, a higher lung compliance allows for a more gradual increase in intrapulmonary pressure, a less abrupt decrease in airflow, and a greater V_T. At normal lung compliance (Panel C), airflow is well maintained throughout the inspiratory cycle, but intrapulmonary pressure rises even more slowly. In a very compliant lung (Panel D), airflow is again well maintained throughout the inspiratory cycle. However, the inspiratory time is too brief to allow for delivery of the full V_T, intrapulmonary pressure never reaches the preset IPAP (15 cm H_2O) [1.5 kPa], and the actual V_T is only slightly greater than in the normally compliant lung. In addition, the highly compliant lung is slow to empty, and auto-PEEP develops with an increase in end-expiratory volume.

Effects of Alterations in Airway Resistance

Figure 6 shows that high airway resistance delays filling of the lung. As expected, V_T delivered through

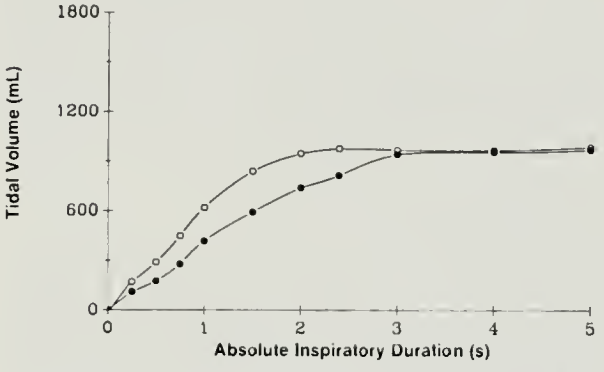

Fig. 6. Tidal volume of the lung model at increasing durations of inspiratory time at 'normal' and high airway resistances. —○— = normal resistance, and —•— = high resistance. EPAP is 5 cm H_2O, PB 10 cm H_2O and lung compliance 0.10 L/cm H_2O. [Refer to box for SI conversion factors.]

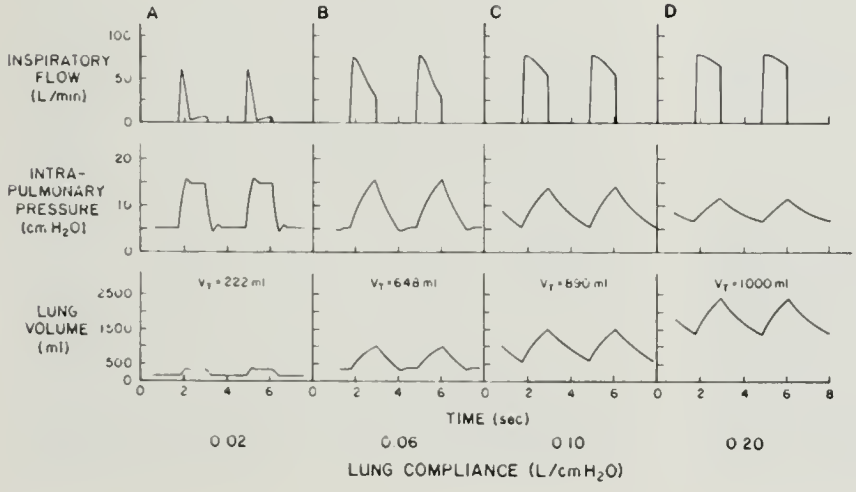

Fig. 5. Simultaneous inspiratory flow, intrapulmonary pressure, and lung volume at lung compliance levels ranging from below normal to above normal. Rate is 20/min, EPAP 5 cm H_2O, %IPAP 40%, and airway resistance 4.6 cm $H_2O \cdot s \cdot L^{-1}$. V_T = tidal volume. [Refer to box for SI conversion factors.]

the high airway resistance eventually reaches that achieved with 'normal' resistance, but there is a delay of roughly 1 second. Thus, high airway resistance will reduce delivered V_T with inspiratory times that would be used clinically—ie, 1-2 seconds.

Results of Evaluation in Patients

Table 1 shows (1) that the BiPAP device was as effective in augmenting minute ventilation as were the standard portable ventilators that the patients had been using, and (2) that the inspiratory pressures generated by the BiPAP device and the standard ventilators were similar. Table 2 shows that in Patients 2, 3, and 4, daytime room-air blood gas/pH values were stable or improved after long-term therapy with the BiPAP device used nocturnally. The patients found that the BiPAP device was as comfortable as their standard portable ventilators, or more so.

Table 1. Ventilatory Values of Patients When Breathing Spontaneously, Using Standard Nocturnal Assistance,* and Using the BiPAP Device

	Patient 1	Patient 2	Patient 3	Patient 4
Spontaneous Breathing				
$\dot{V}_E$ (L/min)	4.4	5.9	10.3	5.0
V_T (mL)	221	368	643	294
f (breaths/min)	20	16	16	17
Standard Nocturnal Assistance				
$\dot{V}_E$	6.5	5.2	14.1	4.9
V_T	295	345	742	288
f	22	15	19	17
PIP (cm H_2O)†	15	10	18	12
BiPAP Trials				
$\dot{V}_E$	6.6	7.0	17.2	6.4
V_T	300	464	905	376
f	22	15	19	17
PIP	15	12	18	18

*Nocturnal assistance refers to standard ventilator used prior to BiPAP trial (Patients 1-3) and to nasal CPAP (Patient 4).

†Refer to box for SI conversion factor.

Abbreviations $\dot{V}_E$ = minute ventilation. V_T = tidal volume, f = breathing frequency, PIP = peak inspiratory pressure (mask pressure).

Discussion

Noninvasive ventilatory support for patients with chronic respiratory failure has been widely used in patients with profound neuromuscular disease such as paralytic poliomyelitis and muscular dystrophy, as well as in patients with kyphoscoliosis.[8] Negative-pressure devices, including the tank respirator and the cuirass, have been successfully employed in the home for such patients, either continuously or nocturnally, for up to 19 years.[9-11] However, negative-pressure devices have a number of limitations, including bulkiness, difficult fitting,[12] patient discomfort,[13] and, perhaps most worrisome, the production of upper-airway obstruction and apneic events during sleep.[1,14] Nasal IPPV avoids many of these problems and has thus far been effective and well tolerated in the several reported series.[1-4]

Our results show that by cyclically delivering two different levels of positive airway pressure, the BiPAP device functions as an effective positive-pressure ventilator and thus appears to be ideal for application in patients requiring only intermittent ventilatory

Table 2. Arterial Blood Gas/pH Values during Three Patients' Spontaneous Breathing of Room Air before and after Nocturnal IPPV and BiPAP Assistance

	pH	P_{aCO_2} (torr)*	P_{aO_2} (torr)*
Patient 2			
Before IPPV	7.37	55	64
After IPPV	7.36	48	72
After BiPAP for 2 months	7.37	40	92
After BiPAP for 4 months	7.39	42	83
Patient 3			
Before IPPV	7.34	76	56
After IPPV	7.45	44	112†
After BiPAP for 2 weeks	7.45	47	107†
After BiPAP for 5 months	7.41	43	105†
Patient 4			
Before IPPV	7.38	56	88
After CPAP for 2 months	7.34	55	77
After BiPAP for 6 weeks	7.39	45	89

*Refer to box for SI conversion factor.

†P_{aO_2} was measured on several occasions in two different laboratories.

assistance via a nose mask. The principles of BiPAP ventilation are similar to those seen with airway pressure-release ventilation (APRV) in that the patient cycles between different levels of positive airway pressure, and spontaneous ventilation between machine-delivered breaths is easily accomplished.[15] However, in BiPAP ventilation the I-E ratio is generally set at < 1, facilitating lung-emptying.

In an artificial lung model, we demonstrated that with normal lung compliance and airway resistance—and commonly used ventilator settings including rates of 12-20/min, %IPAP of 30-40%, EPAP of 2-5 cm H_2O [0.2-0.5 kPa], and PB not exceeding 12-15 cm H_2O [1.2-1.5 kPa]—the BiPAP device functioned as a time-cycled, pressure-limited ventilator. However, maneuvers that further shortened inspiratory time (ie, increased rate or decreased %IPAP) or maneuvers that further prolonged the time necessary to deliver a given V_T (ie, increased PB, high lung compliance, or high airway resistance) introduced a flow limitation. Delivered V_T in these circumstances could be predicted from the product of measured flowrate and inspiratory time, but the preset IPAP level was not reached. Despite this limitation, substantial tidal volumes of > 650 mL could still be delivered, even when high lung compliance was combined with high airway resistance, as would be the case in a patient with severe emphysema. Indeed, the BiPAP device conformed to the equation that Chatburn and Primiano derived to predict V_T delivered by pressure-limited ventilators.[16] There was an excellent correlation ($r = 0.934$, $p < 0.001$) between predicted and actual tidal volumes with the artificial lung, despite variations in compliance, resistance, PB, frequency, and %IPAP.

Our patient trials demonstrated that in a few selected patients, the BiPAP device was as well tolerated and functioned as effectively, even after several months of nocturnal use, as standard portable ventilators. The three patients who requested an extended trial preferred the BiPAP device to their previous systems—one because it has no annoying alarms, one because it weighs less, and one because nasal CPAP alone had failed to effect improvement in either blood gas values or symptoms.

One of our patients had both markedly reduced lung compliance related to severe kyphoscoliosis, and variable airways resistance due to obstructive lung disease. We did not use the device nocturnally in this patient and, in general, would be wary about prescribing a pressure-limited ventilator for patients with unstable airways disease, as their minute volumes may vary with changes in airway resistance. Nevertheless, the BiPAP device had no difficulty in matching the V_T delivered by the standard ventilator and was well tolerated by this patient.

When the device is applied to patients, we recommend that measurements of spontaneous minute ventilation first be obtained with an Ohmeda 5420 volume meter or a similar device. One can then select a machine rate equivalent to the patient's spontaneous breathing rate (or slightly less in tachypneic patients) and set the %IPAP at 30-40%. EPAP is then set at a minimal level to overcome flow resistance within the machine, or higher if the patient is known to have obstructive sleep apnea. Pressure boost is then gradually increased until exhaled minute volume is augmented by 20-30% above spontaneous volume, as tolerated by the patient.

Conclusions

We found the Respironics BiPAP device to be simple to operate, highly portable, and an effective alternative to standard portable ventilators in selected patients with chronic respiratory failure, particularly those receiving nocturnal assisted ventilation via the nasal route. At a cost of about $4,000 it also offers considerable financial advantage over other home ventilators. Because the device has no alarms or backup systems, periodic monitoring of symptoms and arterial blood gases is required, and it should not be used in patients who are ventilator-dependent or who have very limited ventilatory reserve. Also, because it is pressure-limited, the device should be used with caution in patients with high or variable degrees of airway resistance.

PRODUCT SOURCES

Bi-Level CPAP Device:
BiPAP, Respironics Inc, Monroeville PA

Lung Model:
Vent-Aid TTL, Michigan Instruments, Grand Rapids MI

Volume Meter:
Model 5420, Ohmeda, Englewood CO

Pressure Transducer:
Validyne Inc, Northridge CA

Recorder:
Model 1202-9009, Ohmeda, Englewood CO

Standard Portable Ventilators:
Companion 2800, Puritan-Bennett Corp, Carlsbad CA
Thompson-Bantam, Puritan-Bennett Corp, Carlsbad CA
LP-6, Life Products, Boulder CO

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A Continuous-Flow Apparatus for Temporary Inflation of Damaged Endotracheal Tube Cuffs

Glen Tinkoff MD, Eric D Bakow MA RRT, and Raymond W Smith

A ruptured endotracheal (ET) tube cuff is a serious complication in the critically ill patient. Because replacing the faulty tube can be dangerous, we devised a continuous-flow apparatus that can keep a faulty cuff adequately inflated until a more effective airway can be provided in a safe, controlled manner. The apparatus is constructed of commonly available hospital items. It delivers a continuous flow of blended air and oxygen to the ET-tube cuff's pilot balloon line, with flow titrated upward until the cuff leak disappears—usually at a flow of 4 to 6 L/min. A pressure-relief port protects against dangerous overpressurization of the ET-tube cuff, and a system manometer displays and monitors intracuff pressure (= tracheal wall pressure), which should be kept to levels < 25 cm H_2O [2.5 kPa]. We have successfully employed this continuous-flow cuff-inflation system for up to 48 hours in critically ill patients, without encountering complications associated with its use, thus avoiding emergency extubation and 'buying time' until other airway security measures could be safely provided. (Respir Care 1990;35:423-426.)

Introduction

Of the various problems that can occur during long-term endotracheal (ET) intubation,[1] one of the most troublesome is ET-tube failure caused by perforation of the inflatable cuff. This complication usually requires removal of the defective ET tube, followed by re-intubation—sometimes with great risk to the patient. In order to obviate this risk, we developed an apparatus and method for keeping the damaged cuff inflated, making it unnecessary to remove the tube and cuff on an emergency basis. In this paper, we describe the apparatus and report two examples of its use.

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Apparatus

The apparatus, shown in Figure 1, is constructed from materials usually available in hospitals. An air-oxygen blender and standard pressure-compensated

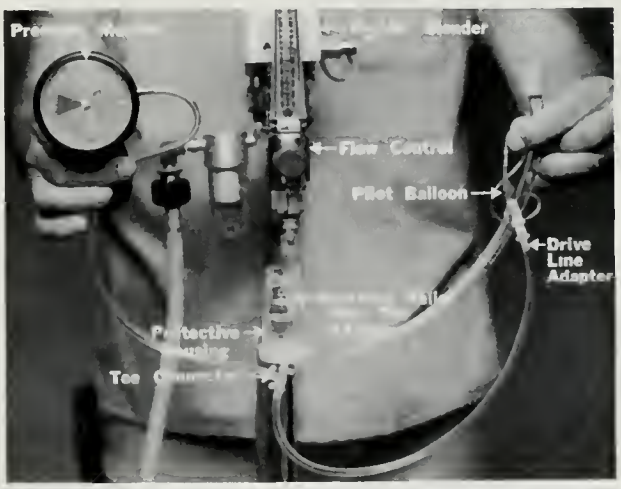

Fig. 1. Components of continuous-flow cuff-inflation apparatus. See text for operation and Table 1 for commercial sources of parts.

Abbreviations Used in This Paper

ET — Endotracheal
F_{IO_2} — Fractional inspired oxygen concentration
PEEP — Positive end-expiratory pressure

A Guide to the Use of SI in This Paper*

The SI unit for pressure is the kilopascal (kPa).
(cm H_2O)(0.098 06) = kPa.

*For further information on SI (le Systemé International d'Unités), see Respir Care 1988;33:861-873 (October 1988) and Respir Care 1989;34:145 (February 1989—Correction).

flowmeter supply blended gas to the cuff-inflating apparatus, which can be secured to the bedside, using I.V. poles or railings. A 4-inch segment of oxygen supply tubing carries blended gas from the flowmeter to a T-piece, the side leg of which remains open to the atmosphere to act as a pressure-relief port. An additional 2 to 3 feet of oxygen supply tubing runs from the bottom leg of the T-piece to a second T-piece. From the side leg of this second T-piece, a length of tubing runs to a manometer, which will provide continuous monitoring of intracuff pressure. From the bottom of the second T-piece, a 1- to 2-foot length of oxygen supply tubing runs to a drive-line adapter, which attaches the apparatus to the pilot balloon valve of the defective cuff.

In operation, continuous flow of blended gas is increased in l-L/min increments until the cuff leak disappears, with the manometer monitoring intracuff pressure (= tracheal wall pressure). The pressure-relief port provided by the open leg of the first T-piece must remain open at all times. Occlusion of this port will lead to excessive pressure and volume in the system and cuff, potentially rupturing the cuff and causing tracheal damage. To make sure that this pressure-relief port will not become occluded, we protect it with a cover fashioned from a disposable plastic vial. This is shown in detail in Figure 2. Table 1 lists the commercial products used in the construction of the apparatus and details how the protective vial is prepared.

We have found that a gas flow of 4 to 6 L/min is usually necessary to maintain safe tracheal wall pressures (< 25 cm H_2O [2.5 kPa]) and to control the leak from the cuff.

Experience

Over the three years prior to the preparation of this report, we used this apparatus several times with success and without untoward incidents. With it, we have been able to maintain cuff inflation up to 48 hours, until a patient could be safely extubated or a tracheotomy performed. Two case summaries will illustrate our experience.

Case #1

A 54-yr-old woman who had been in a motor vehicle accident was admitted to our hospital with a severe closed-head injury, hepatic lacerations, and pelvic fracture with retroperitoneal hemorrhage. She was intubated orotracheally and remained comatose

Fig. 2. Details of pressure-relief port and its protective cover.

after initial resuscitation, surgery, and computed tomography. Because of diffuse cerebral edema, paralyzing agents and mannitol were administered, and she was mechanically hyperventilated.

Over the next 72 hours, the patient had decreasing lung compliance, worsening arterial oxygenation, and radiologic evidence of bilateral diffuse lung infiltrates. On the third hospital day, a leak in the ET-tube cuff was noted. The leak worsened throughout the day, and positive-pressure ventilation became more difficult, with persistent loss of up to half the delivered tidal volume.

Because the patient had massive facial and laryngeal edema, extubation and re-intubation with a new tube and cuff were judged to be extremely hazardous. Therefore, we attached our continuous-flow cuff-inflation system, using a flow of 5 L/min to the cuff so that positive-pressure ventilation could be administered without loss of tidal volume around the ET tube. In this way, we kept the existing tube cuff operating.

Table 1. Parts Used in Continuous-Flow Cuff-Inflation Apparatus

Quantity	Part	Supplier
2	#1115 supply tubing (72″)	Hudson Oxygen 27711 Diaz Rd Box 66 Temecula CA 92390
2	#N-06365-88 T-connector (1/8″ by 1/8″ by 1/8″)	Cole Parmer 7425 N Oak Park Ave Chicago IL 60648
1	#1810 drive line adapter	Baxter Pharmaseal Div 27200 N Tourney Rd Valencia CA 91355
1	#8199 Posey Cuff Flator manometer*	JT Posey Co 6626 W Irving Park Rd Chicago IL 60634
1	#375-238 Accuvett II protective vial (drill 1/8″ hole in each side, 1/4″ hole in lid, 3/8″ hole in bottom; assemble tubing & bleed-off T-piece in vial)	Curtis Mathison 357 Hamburg Turnpike Wayne NJ 07470

*Alternative pressure monitor: #CM high-low pressure alarm, Monaghan Medical Corp, Rt 9, Franklyn Bldg, Plattsburgh NY 12901.

Despite cerebral-protective therapy and monitoring, the patient's neurologic status continued to worsen, and she died of apparent cerebral herniation on the fifth hospital day. The cuff-inflation system was successful in maintaining cuff inflation and safe tracheal wall pressures during the 48 hours it was in use. At autopsy, the ET-tube cuff displayed a 2-mm perforation.

Case #2

A 23-yr-old man was hospitalized after an automobile accident in which he had sustained a closed-head injury, maxillofacial injuries, a pulmonary contusion, and multiple rib fractures. He was nasotracheally intubated for airway control and required tube thoracostomy for hemopneumothorax. Maxillofacial fractures involved the mandible, right zygoma, and infraorbital rim. In the intensive care unit, he remained intubated and was mechanically ventilated because of respiratory compromise. On the fourth hospital day—one day prior to scheduled maxillofacial surgery—a persistent leak in the ET-tube cuff was noted, with substantial loss of tidal volume. A continuous-flow cuff-inflation system was connected to the cuff pilot tube, re-establishing adequate tidal volumes until the patient underwent elective tracheotomy on the following day.

Discussion

Often, loss of gas around an ET-tube cuff is caused by incompetence of the pilot balloon valve; by clamping the connecting tubing or using a three-way stopcock or replacement adapter, this can be simply managed.[2-4] However, management of a leak due to perforation of the cuff itself usually requires removal of the ET tube, which often can pose great risk to the patient. In attempts to obviate such extubation, others have described methods to maintain inflation of a perforated cuff as a stopgap measure until an alternate airway can be secured or the patient can be safely extubated. In 1985, Levack and Scott described using an aneroid manometer, its hand-bulb, an empty 3-liter infusion bag, and two 3-way connectors attached to the pilot balloon tubing.[5] Although the system works, it requires manual inflation, making it somewhat impractical to employ

longer than a few hours. In 1988, Verbough and Camu reported a system that supplied gas to the cuff continuously, but the system lacked pressure monitoring.[6] Also in 1988, Boussard et al described a method for continuous cuff inflation, but their apparatus had no pressure-relief port.[7] Our system overcomes those two shortcomings.

We have successfully tested a bench model of our system with a high-low pressure alarm, but have not had occasion to use an alarm clinically. In our rather limited experience with this apparatus, we have not encountered problems with increases in tidal volume, effects on humidification or PEEP, or changes in F_{IO_2}. In fact, ventilation has consistently been improved. Presumably, the gas flowing through the tear in the cuff enters either the pharynx or the trachea, depending on the location of the hole. We have not had occasion to use this apparatus with a tracheostomy tube.

We emphasize the temporary, stopgap nature of this apparatus. It is certainly not meant to be used in a permanent fashion when an ET-tube cuff is impaired. It should be used only until a more effective airway can be provided in a safe, controlled manner.

ACKNOWLEDGMENTS

We are grateful to Kathleen Moser, Research Department, Lehigh Valley Hospital Center, for her assistance with the text and the illustrations.

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Treatment of Pulmonary Mycobacterial Infections

Hugh S Mathewson MD

Although it no longer can be called the "captain of the men of death" tuberculosis remains an important world health problem. About 8 million new cases are reported annually and 3 million people die of the disease each year.[1] The majority of these cases and deaths occur in undeveloped countries, where poverty, overcrowding, malnutrition, and poor hygiene have long been identified as contributing causes. The prevalence of tuberculosis peaked in Europe and the United States in the early 1800s, when the mines and "dark satanic mills" of the Industrial Revolution provided ample reservoirs of infection, and then progressively declined until the past decade when the incidence began to increase again. The 22,768 new cases reported to the U.S. Centers for Disease Control in 1986 represented a 2.6% increase over the 1985 figure.[2] Since 1986 the numbers have shown a progressive upward trend. About one fourth of new cases are in persons born outside the United States, especially Asian immigrants.[2] Of great concern, however, is the rising incidence in native-born Americans, particularly in the homeless and those confined in penal institutions. About half the new cases are in nonwhite persons.[2] And an important proportion are in drug abusers and those with acquired immunodeficiency syndrome (AIDS).

Professional concern for the recrudescence of tuberculosis has been expressed in some recent publications, notably the September 1989 issue of *Clinics in Chest Medicine*, devoted to mycobacterial diseases,[3] and the Amberson Lecture, given by John F Murray at the 1989 Annual Meeting of the American Thoracic Society.[4] Socioeconomic factors are emphasized, but the lack of antimycobacterial drug development is also pointed out. New drugs for tuberculosis have been few and far between since the introduction of rifampin in 1969. Much of this disinterest appears to stem from the lack of a large potential market as an economic incentive for drug research. At present there are no established drug regimens for the growing numbers of patients with nontuberculous mycobacterial infections. When these infections become disseminated, as has been seen in AIDS victims, little or no benefit may be derived from currently available antimicrobial agents.

Persons with normal defense mechanisms are usually resistant to invasion by mycobacteria. A large component of the resistance is cell-mediated immunity. The patient with AIDS, deprived of T-helper cells, is thus rendered especially susceptible. Nonspecific debilitating factors, such as poor nutrition and poor hygiene, create vulnerabilities that favor bacterial invasion. Mycobacteria are opportunistic and usually of low virulence. Their eradication does, however, require an extended period of drug treatment, usually at least 6 months. This is an epidemiologic problem because homeless or migrant persons or those who fail to comply with a medical regimen may have persisting infectious tuberculosis.

In the patient with active disease, the failure to adhere to a prescribed drug regimen favors the development of resistant organisms. Isoniazid alone will destroy tubercle bacilli by a factor of 10^6; that is, one organism per million will be drug-resistant.[5] Although this may appear to be a minuscule fraction, it is sufficient to promote a drug-resistant strain that many host defenses cannot overcome. Fortunately, the mechanisms of action of isoniazid, rifampin, and ethambutol—the three principal antimycobacterial drugs—are different, and cross-resistance does not occur. Rifampin shows drug-resistance in 1 of 10^9 organisms, ethambutol in 1 of 10^6.[5] The likelihood of an organism being resistant to two agents is the product of their probabilities. Thus, if isoniazid and rifampin are given together, the occurrence of drug-resistant organisms is theoretically $(1 \times 10^8) \times (1 \times 10^6)$, or 1×10^{14}. The consequent substantial reduction in drug-resistant mutants results in greatly improved treatment outcome, but requires that the patient adhere to the drug regimen, and accept the side actions of both drugs. Successful treatment of tuberculosis is currently predicated on the simultaneous use of two or more agents. Patient noncompliance—favoring the development of resistant organisms—is a concern, and new cases of refractory disease are reported in increasing numbers. There is suspicion that some of these cases become treatment failures because the drugs prescribed are either inappropriate or improperly ordered.

Of the three principal antimycobacterial drugs, the mechanism of action of only one, rifampin, is reasonably well understood. Several possibilities have been suggested to explain the action of isoniazid, indicating that none is completely satisfactory. Pharmacology texts are generally silent on the mode of action of

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ethambutol. Fortuitous developments, as exemplified by the discovery of tuberculostatic nicotinamide-like compounds (isoniazid, pyrazinamide, ethionamide) have played a major role in the development of new drugs. Streptomycin, an aminoglycoside, which was the first really effective mycobactericidal agent to be introduced,[6] is still used in drug-resistant cases. The molecular properties of aminoglycosides have been exhaustively explored, but the precise mechanism of their bacteria-killing action is uncertain. Because of their potential for nephro- and ototoxic side actions, streptomycin and its aminoglycoside congeners are not well suited for long-term administration.

Older second-line tuberculostatic drugs (including cycloserine, ethionamide, and aminosalicylic acid) appear to offer less potential as models for new drug development than do antibiotics. With the exception of the 4-quinolone family (ciprofloxacin and others) and clofazimine, virtually no purely synthetic compounds are under study for antimycobacterial activity. By contrast, rifamycin derivatives and other macrolides—semisynthetic congeners of naturally occurring antibiotics—are of greater research interest and clinical promise.

The 4-quinolones were reviewed in this column in 1988.[7] They are synthetic descendants of nalidixic and oxalinic acids, two antimicrobials introduced about 1970. The biochemical target of quinolones is DNA gyrase, a bacterial enzyme involved in a number of critical DNA reactions.[8] The quinolones most effective against mycobacteria are ciprofloxacin and ofloxacin. Only the former is approved for general use in the U.S. Three other compounds are under study as tuberculostatic agents: difloxacin, CI-934, and A-56620.[9] Limited clinical studies indicate that quinolones are likely to be utilized for treatment of tuberculosis, probably in combination with established agents. There is considerable interest in employing them for nontuberculous mycobacterial disease as well. Because their toxic side actions are comparatively mild, quinolones can be given over long periods without undue risk.

Clofazimine is a red dye belonging to the phenazine class. It has been used for several years for treatment of leprosy, always as a component of multiple-drug therapy.[10] Recently it has been employed for complex infections from disseminated *Mycobacterium avium* in patients with AIDS.[9] The drug accumulates readily in tissues, and thus can be given intermittently. It can impart a red color to the skin, but viscerotoxic reactions are uncommon. Its overall clinical usefulness for mycobacterial diseases has not been established because its availability has been limited to use in leprosariums. Other phenazine derivatives are currently under study.[11]

Rifampin is a semisynthetic derivative of rifamycin B, a macrocyclic compound elaborated by *Streptomyces mediterranei*. Rifamycins are complex molecules with structures that have been completely elucidated only within the past 10 years. Rifampin is a broad spectrum antibiotic that inhibits the growth of most gram-positive bacteria and a number of common gram-negative bacilli (*Escherichia coli, Pseudomonas, Proteus*, and *Klebsiella* species). It inhibits DNA-dependent RNA polymerase of mycobacteria and other organisms.[12] The efficacy of rifampin in treatment of *M. tuberculosis* infections is well established. In combination with isoniazid it represents first-line therapy. It is also active against *M. kansasii* and *M. marinum*, but *M. avium, M. fortuitum*, and *M. chelonei* are resistant.[9] The search for rifamycins with broader antimycobacterial spectra is a goal of current drug research.

Rifapentine, a cyclopentyl homologue of rifampin, has recently been prepared commercially, but has not yet been approved by the FDA. It has some pharmacokinetic advantages over rifampin, and probably has greater activity against *M. tuberculosis* and *M. avium*.[13] Rifabutine (ansamycin), a semisynthetic derivative of rifamycin S, is a similar compound with comparable activity.[13]

Macrolide antibiotics, of which erythromycin is the prototype, are large molecular forms elaborated by *Streptomyces* species. They inhibit bacterial protein synthesis by interacting with ribosomal RNA, preventing assembly of new protein. Erythromycin itself has weak antimycobacterial activity, but derivatives have been discovered that are considerably more potent. Roxithromycin (RU-28965) shows marked in-vitro activity against *M. tuberculosis*[14] and *M. avium*.[15] Azithromycin (CP-62933) is a similar compound.[15]

Streptomycin has been mentioned as a second-line antituberculosis drug belonging to the aminoglycoside family. Kanamycin and amikacin have been used in place of streptomycin, and are active in vitro against some nontuberculous species.[16] No new aminoglycosides have thus far been advanced as antimycobacterial agents. Some beta-lactamase resistant cephalosporins have been tried recently, particularly for *M. fortuitum* infections in which cefoxitin appears to be effective.[17] Other chemically unrelated antibiotics (gangamicin,[18] dihydromycoplanecin A[19]) are under study, but no clinical trials have yet been reported.

In summary, tuberculosis appears to be a controllable disease if combinations of first-line agents are taken faithfully over several months. No predictably efficacious drug has yet appeared for disseminated nontuberculous mycobacterial infections. Effective treatment of immunodeficient patients with systemic invasion of these organisms is an unsolved problem of major magnitude.

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Radiographic Findings following Severe Maxillofacial Trauma

Ted R Alber MD and Frederick W Clevenger MD

A 22-year-old man was struck by a car while walking and suffered severe maxillofacial trauma, multiple abrasions and contusions, and an open fracture of the left femur. In the field, emergency medical personnel found the patient apneic and made several unsuccessful attempts to intubate prior to performing an emergency cricothyrotomy. The patient became disoriented and combative during the helicopter flight to the trauma center, but vital signs remained stable. Ringer's lactate (1 L) was infused during flight.

Upon arrival at the trauma center, the patient had a blood pressure of 110/60 and a pulse of 103. Physical exam was remarkable for extensive midface trauma with loose pieces of bone and debris in the oral and nasal cavities. No open chest wounds or active bleeding were noted, but markedly decreased breath sounds were present on the left side. While the admission chest radiograph was being developed (Fig. 1), the patient's hemodynamic status rapidly deteriorated—he became tachycardic, hypotensive, and cyanotic.

Fig. 1. Anteroposterior (AP) chest radiograph obtained shortly after patient's admission to the trauma center, just prior to acute hemodynamic deterioration.

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After appropriate therapy, the patient's hemodynamic status stabilized and a repeat chest radiograph was obtained (Fig. 2).

Fig. 2. Anteroposterior (AP) chest radiograph obtained after appropriate intervention for the life-threatening process present in Figure 1.

Questions

Clinical Changes: What do the clinical changes that occurred while the first radiograph was being developed suggest?

Action or Therapy: What action or therapy is indicated in response to this emergency? Is radiographic documentation needed prior to treatment?

Confirmation: Does Figure 1 confirm the presence of this emergency condition?

Finding: What persistent finding is present in both radiographs?

Action or Therapy: What further action is necessary?

Answers and Discussion on next Page

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Answers

Clinical Changes: The markedly decreased breath sounds present on the left side suggest a pneumothorax. The acute deterioration in hemodynamic status suggests the onset of a tension pneumothorax.

Actions Indicated: The treatment for tension pneumothorax is immediate decompression without a delay for radiographic documentation. In this case, the tension was developing while the first chest radiograph was being performed. Acute decompensation occurred while the film was being developed. The most rapid means of dealing with this life-threatening emergency is decompression by needle catheter, followed by tube thoracotomy. Figure 2 demonstrates a chest radiograph taken after emergency needle decompression, just prior to placement of a chest tube.

Confirmation: The initial radiograph (Fig. 1) reveals a tension pneumothorax with depression of the left hemidiaphragm, free air in the left thoracic cavity (note the outline of the left lung medially), and deviation of the heart and airway structures to the right.

Finding: The casual observer might fail to note the calcified foreign body just to the left of the left heart border overlying the eighth rib (Fig. 1). Note on the follow-up film (Fig. 2) that despite adequate treatment and re-expansion of the lung, the calcified foreign body remains. If you think this foreign body has the outline of a tooth, you are correct.

Actions Indicated: Immediate flexible bronchoscopy was performed, and the tooth was extracted with a bronchoscopic snare (Fig. 3).

Discussion

Booth was the first to report tooth aspiration (in 1953).[1] The patient was a 19-year-old woman who aspirated two lower incisor teeth after a severe mandibular fracture suffered in an automobile crash. The teeth were coughed up by the patient and subsequently passed through the gastrointestinal tract (a process the author followed radiographically). Since then, scattered reports of tooth aspiration have appeared in the literature.[2-8] Although paroxysmal coughing has been reported,[2,6,7] many patients are asymptomatic on presentation.[4-6] If, as in this case, maxillofacial trauma is present, patients may be comatose, and potential signs or symptoms may be masked. Thus, close scrutiny of the chest radiograph becomes critical in establishing the diagnosis. Chest radiographs are always appropriate following maxillofacial trauma, and they warrant careful evaluation.

The patient in this case required an emergency airway as severely injured patients often do. In the unconscious blunt-trauma victim who is apneic, orotracheal intubation with manual cervical-spine immobilization (because of the possibility of neck fracture) is the initial approach. If an airway cannot be secured by this method, cricothyrotomy is performed[9] (as was appropriately done in this case). In the spontaneously breathing unconscious blunt-trauma patient, nasotracheal intubation can be attempted. Clearly, repeated attempts to orotracheally intubate a patient with maxillofacial trauma increases the risk of tooth aspiration.

The suspicion of tooth aspiration increases if the patient presents with loss of consciousness, wheezing, dyspnea, cough, hemoptysis, or chest pain. In cases where the diagnosis is not established early, severe atelectasis, postobstructive pneumonia, and even respiratory failure can develop.[3,10,11] A recent report describes a patient who aspirated denture material during a motorcycle accident and subsequently suffered recurrent pneumonia for 10 years before the foreign body was revealed through bronchoscopy.[12] Although missing teeth would seem to be a prerequisite, McIntosh et al[7] reported aspiration of an

Fig. 3. The tooth extracted from left lower-lobe bronchus, and the bronchoscopic snare used to retrieve the tooth.

unerupted permanent tooth in a pediatric patient with facial trauma.

Treatment of tooth aspiration must be prompt. We found in this case that the flexible bronchoscope provided an adequate view and allowed retrieval of the tooth. Others have found the rigid bronchoscope to be superior, especially in the pediatric age group.[13-16] The rigid bronchoscope is preferred by some because a variety of instruments, including biopsy forceps and Fogarty balloon-tipped catheters, can be passed through the large lumen.[15,17] Although not applicable to our patient, in those who are physically and psychologically able to cooperate, vigorous chest percussion and postural drainage may be an acceptable alternative to bronchoscopy for removal of aspirated foreign bodies, as suggested by Raghu and Pierson.[8] On rare occasions, when endoscopic techniques and chest physiotherapy fail, thoracotomy may be required for bronchotomy.[3,10]

Although common in children, aspiration of foreign bodies is rare in adults. In the present case, a rare occurrence was obscured by an immediate life-threatening co-existent diagnosis. The radiograph taken immediately following decompression of a tension pneumothorax must be scrutinized carefully. One must resist the temptation to only note resolution of the acute process and not to systematically review the entire radiograph. Because of the infrequency of tooth aspiration, the dearth of symptoms, and the associated injuries that can divert the clinician's attention, vigilance must be maintained if this entity is to be recognized.

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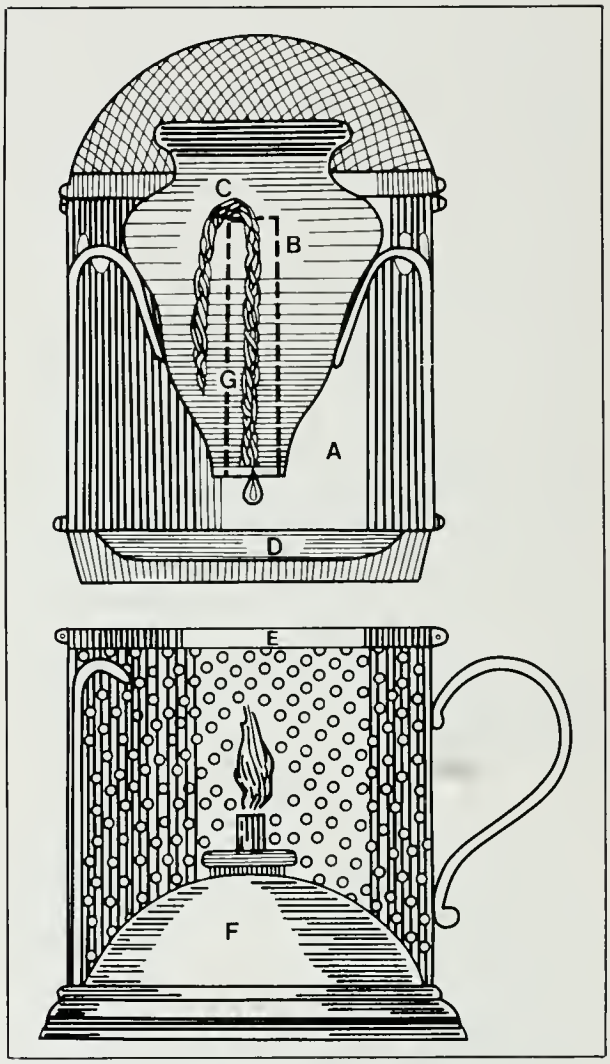
Contributions to this section welcome. Send double-spaced typed material (and illustrations if pertinent) to Historical Notes, RESPIRATORY CARE, 11030 Ables Lane, Dallas TX 75229. Identify source of material and include your name, credentials, and address.

New Inventions in Aid of the Practice of Medicine and Surgery: Patent Vaporiser

We have been furnished by Messrs. Savory and Moore with one of their new vaporisers. It is ingenious in design, simple in construction, and answers the purposes for which it is intended. As will readily be seen by a reference to the engraving, it consists of two parts, separable from one another; the lower containing the lamp, and the upper part a vessel for holding the substance to be vaporised by the aid of a wick, one end of which is inserted into a central dropping tube, and the other immersed in the fluid on the outside of the tube. The liquid carbolic ascends the wick by capillarity, drops on a plate at the bottom, and becomes vaporised by the heat of the lamp placed beneath the plate, when the two parts of the apparatus are adjusted, and the lamp lighted. There can be little doubt that it is an efficient method of diffusing the vapor of disinfectants; and it can be used for purifying the atmosphere of an apartment, or for disinfecting rooms, clothes, bedding, &c. Carbolic acid can thus also be brought into contact with the lungs during respiration, or with any local surface. As to the destructive property exercised by this agent over the atmospheric germs of disease, we are scarcely warranted in expressing an opinion; for we do not know for certain that such germs exist, notwithstanding Profesor Tyndall's so-called "physical demonstration" of their presence and influence; but, for all that, Savory and Moore's Vaporiser is an excellent and very useful little invention.



From The Lancet, September 1870, No. 9, Page 507

This item, originally published nearly 120 years ago, was found and dusted off by David C Lain PhD RRT, formerly of the Medical College of Georgia, now Product Manager for Ohmeda, Columbia, Maryland. Note that the Lancet write-up pronounced the vaporiser "an excellent and very useful little invention" even though "we do not know for certain that [atmospheric germs of disease] exist."

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Respiratory Rehabilitation Program Manual by LaVerne D Harmon BS RNC and J Michael Duncan MD. Notebook, 164 pages. Available from Pulmonary Rehabilitation Consultants, 1147A Professional Drive, Williamsburg VA 23185. 1988. $300.00.

The authors of the **Respiratory Rehabilitation Program Manual** are both affiliated with Williamsburg Community Hospital, Williamsburg, Virginia; Ms Harmon is Director of Cardiopulmonary Rehabilitation and Dr Duncan is Medical Director of Respiratory Rehabilitation. The manual is presented in 10 sections. These sections describe the three phases of the program and include an introduction, quality-assurance information, and a list of reference materials. The first section (the Introduction) contains job descriptions for personnel including program director, exercise physiologist, respiratory care technician, and medical director. I admit to being disappointed that the licensure/credentials listed for the program director were "RN, licensed; certified in BLS and ACLS" (ie, licensed registered nurse, certified in basic and advanced cardiopulmonary life support). I found no job responsibilities that could not be handled by a licensed respiratory therapist with appropriate education, training, and background.

Sections 2 thru 4 deal with Phase I (the inpatient phase) of the rehabilitation program. In Section 2, a review is provided of basic respiratory material to be presented to participants prior to their discharge from the hospital. This section identifies objectives for these educational sessions. In Section 3, handouts with basic information on medications are provided. Section 4 contains a patient flow sheet that is basically a check-off sheet for patient education sessions.

Sections 5 and 6 deal with Phase II of the rehabilitation program. Section 5 begins with a definition of pulmonary rehabilitation and a list of overall objectives for the program. A suggested method of program implementation, along with referral sources, admission criteria, and guidelines for admission are included. It might have been appropriate to have placed these earlier in the manual because they serve as an introduction to rehabilitation. Procedures, protocols, and policies are also covered in Section 5. The exercise procedure guidelines provided offer a good base for developing an exercise-testing protocol. Billing procedures for rehabilitation programs are briefly discussed, and revenue codes and current procedural terminology (CPT) codes are listed for commonly used therapies. Section 5 concludes with lecture outlines on benefits of exercise, medications, nutrition, and psychosocial aspects of COPD. A list of suggested teaching materials is also included.

Charting forms and patient instruction sheets for testing procedures are provided in Section 6. The Cardiopulmonary Exercise Test Worksheet, and the Assessment and Program Evaluation forms are excellent. The Patient Self-Assessment and Quality of Life Scale forms are designed to reveal valuable information about rehabilitation participants. Purchase of the book bestows the right to copy, which means that all forms may be duplicated and distributed.

Sections 7 and 8 deal with Phase III (the maintenance phase) of the rehabilitation program. Provided in these sections are instructions for home exercise and a 'metabolic activity range' chart.

In Section 9, a solid base is provided for developing a quality assurance plan. In Section 10, a list of pulmonary rehabilitation references is supplied.

Many of the charting and assessment forms in the manual are excellent. However, the inpatient education material provided in Section 2 for the instructor's use is extremely basic, and the patient handouts provided in Section 3 do not address anatomy and physiology, disease processes, postural drainage and percussion, nutrition, stress management, body mechanics, breathing control techniques and exercises, or asthma attack management. Anyone teaching this portion of the program should have a thorough understanding of these subjects and be able to augment the materials provided so that the information can be presented at an appropriate level to the patient. Information on reimbursement, as provided by various insurance companies, would have been a helpful addition to Section 5.

Even though the $300.00 cost is high, the **Respiratory Rehabilitation Program Manual** provides some good basic information that will be useful for those who are considering developing a rehabilitation program for pulmonary patients.

Lori L Kondas CRTT CPFT
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Master Guide for Passing the Respiratory Care Credentialing Exams, 2nd ed, by Terrance M Krider BS RRT, Rick Meyer BS RRT, and William A Syvertsen MS RRT. Softcover, 457 pages. Claremont CA: Education Resource Consortium Inc, 1989. $29.95. To order, contact: ERC Inc, 190 E Sweetbriar Dr, Claremont CA 91711. (714) 621-6261.

Adequate preparation for entry-level certification (CRTT) and advanced-

level registration (RRT) with the National Board for Respiratory Care (NBRC) is and has been the desire of all of us aspiring to master the profession of respiratory care. Such preparation is 'easier said than done,' and requires a positive mental attitude that comes from internal motivation. If you feel motivated to become credentialed in respiratory care (Certified or Registered), or if you consider yourself a facilitator of others so motivated, then we highly recommend **Master Guide for Passing the Respiratory Care Credentialing Exams.**

As department heads of both entry-level (CRTT) and advanced-level (RRT) programs, we have in the past leaned heavily on other review texts as well, including **Respiratory Care: National Board Review,** by CA Brainard (Respir Care 1985;30:134); **Comprehensive Review in Respiratory Care,** 2nd ed, by MV Wojciechowski and E Neff (Respir Care 1989;34:42); and **Clinical Simulations for Respiratory Care,** by RW Beckham (Respir Care 1980;25:286).

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Master Guide is a welcome addition to these and other respiratory care texts.

The authors are well qualified to write such a credentialing review (Program Director, Clinical Coordinator, and Administrative Assistant in California). They focus on the three main areas assessed in the national exams for both certification and registry: Data, Equipment, and Therapy. They not only address the latest examination matrices (published by the NBRC in 1987) but also include additional improvements over the first edition (1984), including a subdivision of individual sections into separate chapters and expanded to accommodate the new NBRC exam matrix. The discussions of pathology (Chapter 10) have been expanded to include pneumoconiosis, postoperative pulmonary complications, and AIDS (as part of pneumonia). An additional 150 self-assessment questions are provided and the entire self-assessment (300 questions) is now consolidated into one easy-to-use section. We especially like the annotated reference at the end of each section that furnishes a descriptive up-to-date review of other sources of information for those wishing a more comprehensive discussion of key topics.

Master Guide is a valued addition to the armamentarium required by educators whose curriculums need to meet national guidelines and whose graduates need to show successful outcomes on the Board exams. We especially found Chapter 1, "Improving Your Results on the Credentialing Exams," to be helpful. In this chapter both the entry-level certification (CRTT) and the advanced-level registry (RRT) examinations are well described. More importantly, the authors make a concerted attempt to psychologically prepare the reader for success instead of failure. We especially found joining the "5 Percent Club" to be unique and helpful in developing the positive attitudes necessary for success on the credentialing exams.

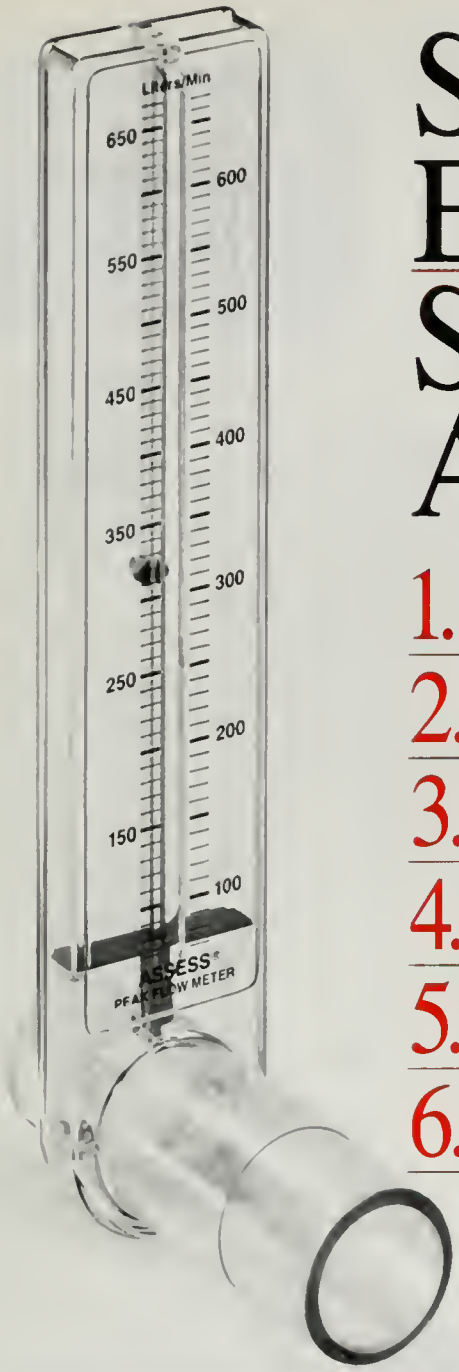
We have alluded to what we consider to be the strengths of **Master Guide** and must admit to finding what we consider a problem and a weakness as well (don't they exist in all texts?!). The problem has been to find **Master Guide** on the bookshelves of the local bookstore! We sought the first edition in 1984, and tried to find the second edition in 1990! The ordering information provided in this review should make this valuable book available to all who seek it. The weakness is that while the content seems to be of great value for the written credentialing exams (both entry and advanced level), it seems of little value in adequately preparing individuals for the clinical simulation exams. We would have liked to see some latent image content/practice in **Master Guide**—which of course would make it more expensive as well.

In conclusion, it is important to note that Krider, Meyer, and Syvertsen are the first authors to our knowledge to actually reflect and discuss the specific content areas that are presently assessed by the NBRC in the certification and registry credentialing exams. The softcover second edition of **Master Guide** is an excellent, up-to-date addition to the library of anyone who wishes to become credentialed in respiratory care and anyone who helps others become so credentialed. It can also be used by practicing respiratory care practitioners to assess their strengths and weaknesses—something we must all do from time to time. We enjoyed reviewing this very fine text, and look forward to all of our graduates mastering the content found within its soft covers as well.

Mimi Casey Bartel MEd RRT
Department Head
Respiratory Care Technician Program
(Entry-Level)

Ralph E Bartel MEd RRT
Department Head
Respiratory Therapist Program
(Advanced-Level)

Houston Community College System
Houston, Texas



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References 1. Hendler J, Shapiro S, Ogirala R, Aldrich T. Accuracy of new and used portable peak flow meters. Abstract presented at the 46th Annual Meeting of the American College of Allergy and Immunology, Orlando, FL, November 11-15, 1989. 2. Shim CS, Williams MH. Relationship of wheezing to the severity of obstruction in asthma. *Arch Intern Med 143* 890, 1983. 3. Williams MH. Expiratory flow rates. Their role in asthma therapy. *Hospital Practice* 95, October, 1982. 4. Woolcock AJ, Yan K, Salome CM. Effect of therapy on bronchial hyperresponsiveness in the long-term management of asthma. *Clinical Allergy 18* 165, 1988.

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In June .....

- Noninvasive Monitoring: Present, Past & Future
- What Makes Noninvasive Monitoring Tick?
- Instrument Error and Method Agreement
- Monitoring without Machinery
- Capnography: Instrumentation & Clinical Application
- Transcutaneous Monitoring: Instrument & Clinical Application
- Pulse Oximetry: Instrumentation & Clinical Application

Not-for-profit organizations are offered a free advertisement of up to eight lines to appear, on a space available basis, in Calendar of Events in RESPIRATORY CARE. Ads for other meetings are priced at $5.50 per line and require an insertion order. Deadline is the 20th of month two months preceding the month you wish the ad to run. Submit copy and insertion orders to: Calendar of Events, RESPIRATORY CARE, 11030 Ables Lane, Dallas TX 75229

Calendar of Events

AARC & AFFILIATES

May 16-18 in Cincinnati, Ohio. The Indiana, Kentucky, and Ohio Societies for Respiratory Care host the 17th Annual Region II Conference at the Albert B Sabin Convention Center and the Clarion Hotel. Mechanical ventilation is the primary topic. Contact David Dunlap at (606) 292-4271.

May 16-18 in El Paso, Texas. The Southwest Region Texas Society for Respiratory Care presents its 19th Annual Conference "Into the 90s" at the El Paso Airport Hilton. Contact Patsy Barrazza, Thomason Hospital, Respiratory Care Department, 4815 Alameda, El Paso TX 79905. (915) 544-1200, ext. 110.

May 26-29 in Saint John, New Brunswick, Canada. The Canadian Society of Respiratory Therapy presents its 25th Annual Educational Forum and Annual Meeting at the Saint John Trade and Convention Centre. Contact John Unrau, Box 676, Hampton, New Brunswick, Canada E0G 1Z0. (506) 648-6704.

May 29-June 2 in Daytona Beach, Florida. The Florida Society for Respiratory Care (FSRC) presents "Sunshine Seminar 1990" at the Daytona Beach Marriott Hotel. Contact FSRC, PO Box 65, Hobe Sound FL 33475-0065.

June 6-8 in Grand Island, Nebraska. The Nebraska Society for Respiratory Care presents its Great Plains Conference on Respiratory Care at the Midtown Holiday Inn. Contact Bruce Couillard RRT at (402) 390-4677.

June 7-8 in Kansas City, Kansas. The Kansas Respiratory Care Society presents its 13th Annual Education Seminar at the Overland Park Marriott. Speakers include Cheryl Brown and Dean Hess. Contact Frank Hart at (913) 676-2175.

June 7-8 in Syracuse, New York. The Central New York Chapter of the New York State Society for Respiratory Care presents its 22nd Annual Seminar, "Respiratory Care: Concepts for the '90s." Thursday, June 7—an afternoon golf tournament is followed by Sputum Bowl competition and a welcome party. Friday, June 8—meeting topics include positive-pressure mask ventilation, aerosol devices and deposition, inverse-ratio ventilation in adults, emergency management of asthma in children, and future directions in respiratory care. Contact Claire Aloan RRT, (315) 478-5920; or Joe Kieffer RRT, (315) 425-7572.

June 8-9 in Charlottesville, Virginia. The Virginia Society for Respiratory Care sponsors an entry-level case study workshop at Piedmont Virginia Community College. Contact Leslee Smith, Northern Virginia Community College, 8333 Little River Turnpike, Annandale VA 22003.

June 12-14 in Scottsdale, Arizona. The Arizona Society for Respiratory Care presents its Annual Education Seminar at the Wyndham Paradise Valley Resort. This year's theme is "Many Roles, Common Goals." Featured speakers are Irwin Ziment MD, Karen Larson RRT, Katherine Chavigny MSN RN PhD, David Walker MA RRT, Jerome Sullivan MS RRT (AARC president), Jack Wanger MBA RRT RPFT, and Neil McIntyre MD. Topics include MDI, patient management in flight, sleep apnea, and laser surgery. Contact Karen Staudenmier RRT at (602) 345-7777.

June 13-15 in Roundtop, New York. The New York and New Jersey Societies for Respiratory Care host the 2nd Annual Managers and Educators Conference at the Winter Clove Inn. Contact Patty Bowe, (518) 270-7454 or Ken Wyka, (201) 456-5503.

June 13-15 in St Charles, Illinois. The Illinois Society for Respiratory Care presents its annual convention, "Westward Ho: Golden Opportunities in Respiratory Care," at Pheasant Run. Contact Trudy Watson RRT, Black Hawk College, 6600 34th Ave, Moline IL 61265, (309) 796-1311, ext 3303; or Vince Madama RRT, Rock Valley College, 3301 N Mulford Dr, Rockford IL 61103, (815) 654-4413 or (815) 654-4410.

June 14-16 in San Diego, California. The California Society for Respiratory Care (CSRC) presents the 1990 Annual Convention, "Pursuing Professional Excellence," at the Sheraton Harbor Island East in San Diego. Contact CSRC, 24307 Magic Mountain Parkway, Suite 288, Valencia CA 91355. (805) 298-4010.

July 30-31 in Reno, Nevada. The Nevada Society for Respiratory Care presents its 4th Annual Symposium at the Flamingo Hilton. Contact Kathy Green RRT, PO Box 2168, Carson City NV 89702. (702) 882-1361, ext 258.

August 24-26 in Marco Island, Florida. The Florida Society for Respiratory Care presents the Southernmost Sandcastle Seminar at the Marco Beach Hilton Resort. Seminar fees cover all Saturday meals including a sunset buffet beach party. Bring your own musical instruments to join our sing-along or win a prize for the "best sandcastle on the beach." Ample time is planned for exhibits. Deadline for reservations is July 12. Contact Dave Robbins RRT, Coral Gables Hospital, 3100 Douglas Rd, Coral Gables FL 33133. (305) 441-6819.

OTHER MEETINGS

April 20-June 24. Primedica offers preparatory programs for the NBRC examinations in the following cities: Registry Review—Baltimore, Maryland, and Dallas, Texas; Entry-Level Review—Atlanta, Georgia, and Dallas, Texas. Contact Sandy Blair, Primedica, 1841 West Oak Parkway, Suite C, Marietta GA 30062. (800) 647-3729, ext 3139; or (404) 426-0861, ext 3139.

June 8-10 in Memphis, Tennessee. St Joseph Hospital sponsors the 10th Annual Respiratory Care Symposium. The program provides a review of the basics of respiratory care and prepares candidates for the certification examination. Contact Debby Spray RRT or Dorothy Younginer RRT at St Joseph Hospital, Respiratory Care Services, 220 Overton Ave, PO Box 178, Memphis TN 38101, (901) 577-2780.

March 10-13, 1991, in Denver, Colorado. The National Jewish Center for Immunology and Respiratory Medicine sponsors the 3rd International Conference on Pulmonary Rehabilitation and Home Mechanical Ventilation at the Denver Hyatt. Workshop topics are home ventilator care and pulmonary rehabilitation. Program chairman is Barry Make MD. Contact Adele Gelfand, Conference Coordinator, at (303) 398-1359.

Notices

Notices of competitions, scholarships, fellowships, examination dates, new education programs, and the like will be listed here free of charge. Items for the Notices section must reach the Journal 60 days before the desired month of publication (January 1 for the March issue, February 1 for the April issue, etc). Include all pertinent information and mail notice to RESPIRATORY CARE Notices Dept, 11030 Ables Lane, Dallas TX 75229.

ARCF Literary Award

- The American Respiratory Care Foundation announces a $1000 Literary Award—funded by Radiometer America Inc—for the best case report published in RESPIRATORY CARE from October 1989-December 1990. The winner will be announced on December 8, 1990, at the AARC Annual Meeting, and in the January 1991 issue of RESPIRATORY CARE. All case reports will be considered for the award, and no application is necessary.

AARC SUMMER FORUM

The Registry Resort, Naples, Florida, July 13-15, 1990
The Westin, Vail, Colorado, July 11-13, 1991

AARC ANNUAL CONVENTION SITES & DATES

1990—New Orleans, Louisiana, December 8-11
1991—Atlanta, Georgia, December 7-10
1992—San Antonio, Texas, December 12-15
1993—Nashville, Tennessee, December 11-14
1994 Las Vegas, Nevada, December 12-15
1995—Orlando, Florida, December 2-5

THE NATIONAL BOARD FOR RESPIRATORY CARE

1990 Examination and Fee Schedule

CRTT Examination

EXAMINATION DATE:	JULY 21, 1990
Applications Accepted Beginning:	March 1, 1990
Application Deadline:	May 1, 1990
EXAMINATION DATE:	NOVEMBER 10, 1990
Applications Accepted Beginning:	July 1, 1990
Application Deadline:	September 1, 1990

RRT Examination

EXAMINATION DATE:	JUNE 2, 1990
Applications Accepted Beginning:	December 1, 1989
Application Deadline:	February 1, 1990
EXAMINATION DATE:	DECEMBER 1, 1990
Applications Accepted Beginning:	June 1, 1990
Application Deadline:	August 1, 1990

CPFT Examination

EXAMINATION DATE:	JUNE 2, 1990
Applications Accepted Beginning:	December 1, 1989
Application Deadline:	April 1, 1990

RPFT Examination

EXAMINATION DATE:	DECEMBER 1, 1990
Applications Accepted Beginning:	July 1, 1990
Application Deadline:	September 1, 1990

Fee Schedule

Entry Level CRTT—new applicant:	$ 75.00
Entry Level CRTT—reapplicant:	$ 50.00
Combined RRT Written and Clinical Simulation:	$175.00
Written Registry Only—new applicant:	$ 75.00
Written Registry Only—reapplicant:	$ 50.00
Clinical Simulation Only—new or reapplicant:	$100.00
Entry Level CPFT—new applicant:	$100.00
Entry Level CPFT—reapplicant:	$ 80.00
Advanced RPFT—new applicant:	$150.00
Advanced RPFT—reapplicant:	$130.00
CRTT Recredentialing:	$ 25.00
RRT Recredentialing:	
Written Registry Examination	$ 25.00
Clinical Simulation Examination	$ 65.00
CPFT Recredentialing:	$ 25.00
RPFT Recredentialing:	$ 90.00
Membership Renewal CRTT/RRT/CPFT/RPFT	$ 12.00
Membership Renewal Combination of CRTT/RRT and CPFT/RPFT	$ 18.00

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RESPIRATORY CARE
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The American Association for Respiratory Care and its science journal, RESPIRATORY CARE, invite submission of brief abstracts related to any aspect of cardiorespiratory care. The abstracts will be reviewed, and selected authors will be invited to present papers at the Open Forum during the AARC Annual Meeting in New Orleans, Louisiana, December 8-11, 1990. Accepted abstracts will be published in the November 1990 issue of RESPIRATORY CARE. Membership in the AARC is not necessary for participation.

Specifications

Please Read Carefully

An abstract may report (1) an **original study**, (2) the **evaluation of a method or device**, or (3) a **case or case series**. Topics may be aspects of adult acute care, continuing care/rehabilitation, perinatology/pediatrics, cardiopulmonary technology, health occupations education, or management of personnel and health-care delivery. The abstract may have been presented previously at a local or regional—but not national—meeting and should not have been published previously in a national journal.

The abstract will be the only evidence by which the reviewers will decide whether the author should be invited to present a paper at the Open Forum. Therefore, *the abstract must provide all important data, findings, and conclusions.* Give specific information. Do not write such general statements as "Results will be presented" or "Significance will be discussed."

Essential Content Elements

An **original study** abstract ***must*** include (1) Introduction: statement of research problem, question, or hypothesis; (2) Method: description of research design and conduct in sufficient detail to permit judgment of validity; (3) Results: statement of research findings with quantitative data and statistical analysis; (4) Conclusions: interpretation of the meaning of the results.

A **method/device evaluation** abstract ***must*** include (1) Introduction: identification of the method or device and its intended function; (2) Method: description of the evaluation in sufficient detail to permit judgment of its objectivity and validity; (3) Results: findings of the evaluation; (4) Experience: summary of the author's practical experience ***or*** a notation of lack of experience; (5) Conclusions: interpretation of the evaluation and experience. Cost comparisons should be included where possible and appropriate.

A **case report** abstract ***must*** report a case that is uncommon or of exceptional teaching/learning value and must include: (1) case summary and (2) significance of case. Content should reflect results of literature review. The author(s) should have been actively involved in the case and a case-managing physician must be a co-author or must approve the report.

Abstract Format and Typing Instructions

An optical scanner will be used to process abstracts. ***Do not use a dot-matrix printer.*** First line of abstract should be the title. Title should explain content. Type the abstract ***double-spaced*** on ***plain white bond paper***, on ***one page*** only (copier bond is excellent). Do not underline or boldface and insert only one letter space between sentences. Provide a 1-inch margin top and bottom, a 1/2 inch left margin, and an approximate 1/2 inch ragged right margin.

No identification of authors or institutions is to appear on the abstract sheet or in the abstract itself. Make the abstract all one paragraph. Data may be submitted in table form ***provided the table width is limited to 60 letter spaces*** (ie, letters or numbers plus necessary blank spaces = 60). ***No*** figures or illustrations are to be attached to the abstract.

Type all information required to complete the author information form on the other side of this page. A photocopy of good quality may be used.

Standard abbreviations may be employed without explanation. A new or infrequently used abbreviation should be preceded by the spelled-out term the first time it is used. Any recurring phrase or expression may be abbreviated if it is first explained.

Check the abstract for (1) errors in spelling, grammar, facts and figures; (2) clarity of language; (3) conformance to these specifications. An abstract not prepared as requested may not be reviewed.

Questions about abstract preparation may be telephoned to the editorial staff of RESPIRATORY CARE at (214) 243-2272.

Deadlines

The mandatory Final Deadline is June 9 (postmark). Authors will be notified of acceptance or rejection ***by letter only*** — to be mailed by August 15.

Authors may choose to submit abstracts early. Abstracts received by March 26 will be reviewed and the authors notified by May 1. Rejected abstracts will be accompanied by a written critique that should in many cases enable authors to revise their abstracts and resubmit them by the final deadline (June 9).

Mailing Instructions

Mail 1 copy of the abstract, 1 author information sheet, and a stamped, self-addressed postcard (for notice of receipt) to:

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RESPIRATORY CARE

Instructions for Authors and Typists

These Instructions are meant to guide authors and typists, including veterans in those roles, in the production of quality manuscripts. Perfection is not expected, but the well-prepared manuscript has the best chance for prompt review and early publication.

General Requirements

Submissions should (1) be related to respiratory care, (2) be planned for one of the publication categories below, and (3) be prepared as indicated in these Instructions. A letter accompanying the manuscript must specify the intended publication category, be signed by all the authors, and, when there are two or more authors, state that "We, the undersigned, have all participated in the work reported, read the accompanying manuscript, and approved its submission for publication."

Publication Categories

Research Article (Study): A report of an original investigation.
Evaluation of a Device/Method/Technique: A description and evaluation of an old or new device, method, technique, or modification.
Case Report: A report of a clinical case that is uncommon or of exceptional teaching value. The author(s) must have been associated with the case. A case-managing physician must be one of the authors or, if not an author, must supply a letter approving the manuscript.
Case Series: Like a Case Report but including a number of cases.
Review Article: A comprehensive, critical review of the literature and state of the art of a pertinent topic that has been the subject of 40 or more published research papers.
Overview: A critical review of a pertinent topic about which not enough research has been published to merit a Review Article.
Update: A report of subsequent developments in a topic that has been critically reviewed (not necessarily in this journal).
Point of View: A paper expressing the author's personal opinions on a pertinent topic.
Special Article: If a paper does not fit one of the foregoing categories but is pertinent, the editors may consider it as a Special Article.
Editorial: A paper that draws attention to a pertinent concern.
Letter: A signed communication about material published in this journal or on topics of interest or value to readers.
Blood Gas Corner: A brief, instructive case report (real or fictional) involving invasively or noninvasively obtained respiratory care blood data, followed by questions for readers—with answers and discussion.
PFT Corner: Like Blood Gas Corner but involving pulmonary function testing.
Test Your Radiologic Skill: Like Blood Gas Corner and PFT Corner but involving pulmonary-medicine radiography and including one or two 4 × 5 or 5 × 7 inch prints of radiographs. The case must be real.
Review of Book, Film, Tape, or Software: Anyone interested in writing a review can discuss it with an editor.

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Authors are urged to obtain the RESPIRATORY CARE Author's & Typist's Kit. The Kit provides *authors* with specific guidance about writing a research paper, writing a case report, converting to and from SI units, and in-house manuscript review. *Typists* can use the Kit's Model Manuscript, a list of journal name abbreviations, and a copy of these Instructions. The Kit is free from the Journal office.

Preparing the Manuscript

General Concerns—*Typist*

- Double-space ALL lines, including those in references, figure legends, and tables. Do not justify right margins.
- Number pages in upper right corner and leave margins of 1½" or more on all four sides of the page.
- For research articles, follow format of Model Manuscript, Respir Care 1984;29:182 (Feb 1984).
- Meticulously follow instructions for typing references.

General Concerns—*Author:*

- Structure manuscript as specified hereafter.
- Provide all requested information on title page as specified hereafter.
- Proofread manuscript for completeness, clarity, grammar, spelling; be sure all references, figures, and tables are cited in the text.
- Consider having paper reviewed in-house before submission.
- Have all co-authors proofread and approve manuscript and sign submission letter.

Manuscript Structure

Most kinds of papers have standard parts in a standard order. However, papers can vary individually, and not every paper will have all the parts listed here.

Research Article: Title page, abstract page, continuous text (Introduction, Materials & Methods, Results, Discussion), Product Sources page, Acknowledgments page, references, tables, figure legends. Please consult "Writing a Research Paper," Respir Care 1985;30:1057 (Dec 1985) and Model Manuscript, Respir Care 1984;29:182 (Feb 1984).
Evaluation of Device/Method/Technique: Title page, abstract page, continuous text (Introduction, Description of Device/Method/Technique, Methods of Evaluation, Results of Evaluation, Discussion), Product Sources page, Acknowledgments page, references, tables, figure legends.
Case Report or Case Series: Title page, abstract page, continuous text (Introduction, Case Summary, Discussion), Acknowledgments page, references, tables, figure legends. Also see "How To Write a Better Case Report," Respir Care 1982;27:29 (Jan 1982).
Review Article: Title page, Table of Contents page, continuous text (Introduction, History, Review of Literature, State of the Art, Discussion, Summary), references. May include figures & tables. No abstract. Table of Contents optional. Other formats may be appropriate.
Overview, Update, Point of View, or Special Article: Title page, text (introduction, message), references, tables, figure legends. No abstract.
Letter: Title page (provide a title), text, writer's name & affiliation, references. Tables & figures may be included. Double-space everything. Write "For Publication" on title page.

Structure: Important Details

Title Page: List title of paper, all authors' full names, degrees, credential letters, professional positions, and affiliations. List correspondence address, telephone number, and reprint address if desired. Name sources of grants or other support. Identify any author's consulting or commercial relationships that pertain to the paper's topic.

Abstract Page: Number this Page 1. List paper's title but omit authors' names. Abstract should be 200 words or less and must be informative, briefly specifying main points of paper, such as methods, results, and conclusions drawn.

Statistical Analysis: In research articles, identify statistical tests and chosen level of significance in the Methods section. In Results section, report actual P values.

Figures (illustrations): All photographs, diagrams, & graphs must be numbered as Figure 1, Figure 2, etc, according to the order in which each is first mentioned in the text. Photographs must be glossy prints 5 × 7 to 8 × 10 inches and should be black & white unless color is essential. Letters and numerals must be neat and large enough to remain legible if figure is reduced in size for publication. Final figures must be of professional quality, but 'rough' sketches may accompany the submitted manuscript, with final figures to be prepared after review. Identify each figure on back with a stick-on label showing figure number and arrow indicating top; omit author's name. Cover label with clear tape so ink will not smudge other prints. Supply three sets of unmounted figures. If figure has been published before, include copyright-holder's written permission to use it.

Figure Legends: List figure legends on a separate page, not on figures. If a figure has been published before, list the source in the legend.

Tables: Type each table on a separate page. Avoid more than 8 columns across. Continue a deep table on following pages. Give each table a number and descriptive title, placed above the table. Double-space ALL lines in tables, including column headings and footnotes.

Drugs: Brand names may be given, but always also show generic names.

Units of Measurement: In addition to conventional units of measure, show SI values and units in brackets after conventional expressions: ie, "PEEP, 10 cm H_2O [0.981 kPa]." For conversion to SI, see RESPIRATORY CARE 1988;33:861-873 (Oct 1988).

Commercial Products: If three or fewer commercial products are named in the text, list the manufacturer's name and location in parentheses the first time each is mentioned. If four or more products are named, do not list manufacturers in the text; instead, name the products and manufacturers in a Products Sources list at the end of the text. Provide model numbers when available.

Abbreviations: Use an abbreviation only if the term occurs several times in the paper. Write out the full term the first time it appears, followed by the abbreviation in parentheses. Thereafter, employ the abbreviation alone. Never use an abbreviation without defining it. Do not create new abbreviations unless absolutely necessary.

References:

- Use references to support statements of fact, indicate sources of information, or guide readers to further pertinent literature.
- Cite only published works—or works accepted for publication. When listing an accepted but still unpublished work, designate the accepting journal's name, followed by "(in press)."
- In the text, cite references by superscript numerals (half space above text), not in parentheses. The first reference cited in the text is number 1, the next is number 2, etc.
- In the reference list, place the cited works in numerical order.
- For the reference list, obtain author names, article and book titles, dates, volume and page numbers from the *original* cited articles and books, not from secondary sources such as other articles' reference lists, which often are inaccurate.
- Type references in medical-journal style. Examples appear at the end of these Instructions. Abbreviate journal names as in *Index Medicus*. A list of many journal-name abbreviations was published in Respir Care 1988;33:1050 (Nov 1988).
- DOUBLE-SPACE the lines of references.
- List ALL authors' names. Do not use "et al" to substitute for names.
- Identify abstracts, editorials, and letters as such. See examples.

Personal Communications, Unpublished Papers, and Unpublished Observations: List unpublished items in parentheses in the text, not in the reference list.

Examples of How To Type References

Notes: Although the examples here are printed with single-spaced lines, please double-space references in manuscripts. Also, note that words in article and book titles are not capitalized except proper names.

Standard Journal Article:

1. Shepherd KE, Johnson DC. Bronchodilator testing: An analysis of paradoxical responses. Respir Care 1988;33:667-671.

Corporate Author Journal Article:

2. American Association for Respiratory Care. Criteria for establishing units for chronic ventilator-dependent patients in hospitals. Respir Care 1988;33:1044-1046.

Article in Journal Supplement:

(Journals differ in their methods of numbering and identifying supplements. Supply sufficient information to allow retrieval.)

3. Reynolds HY. Idiopathic interstitial pulmonary fibrosis. Chest 1986;89(3, suppl):139s-143s.

Abstract in Journal:

(Abstracts are not strong references; when possible, full papers should be cited. When cited, abstracts should be identified as such.)

4. Lippard DL, Myers TF, Kahn SE. Accuracy of pulse oximetry in severely hypoxic infants (abstract). Respir Care 1988;33:886.

Editorial in Journal:

5. Rochester DF. Does respiratory muscle rest relieve fatigue or incipent fatigue? (editorial). Am Rev Respir Dis 1988;138:516-517.

Letter in Journal:

6. Smith DE, Herd D, Gazzard BG. Reversible bronchoconstriction with nebulised pentamidine (letter). Lancet 1988;2:905.

Personal Author Book:

7. Nunn JF. Applied respiratory physiology. New York: Appleton-Century-Crofts, 1969.

Note: To specify pages cited in a book, place a colon after the year and then list the page(s). Examples: 1969:85 (one page), 1963:85-95 (series of contiguous pages), 1963:85,95 (separated pages).

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8. American Medical Association Department of Drugs. AMA drug evaluations, 3rd ed. Littleton CO: Publishing Sciences Group, 1977.

Book with Editor, Compiler, or Chairman as 'Author':

9. Guenter CA, Welch MH, eds. Pulmonary medicine. Philadelphia: JB Lippincott, 1977.

Chapter in Book:

10. Pierce AK. Acute respiratory failure. In: Guenter CA, Welch MH, eds. Pulmonary medicine. Philadelphia: JB Lippincott, 1977:171-223.

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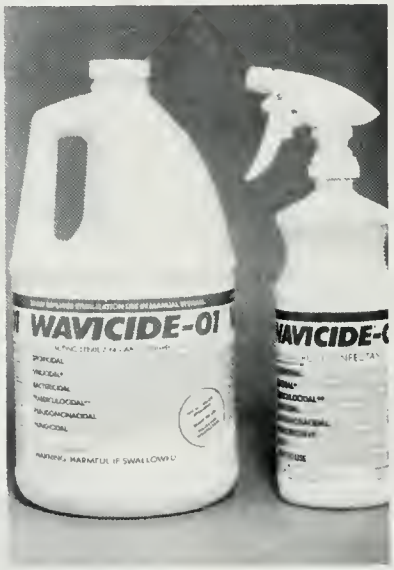

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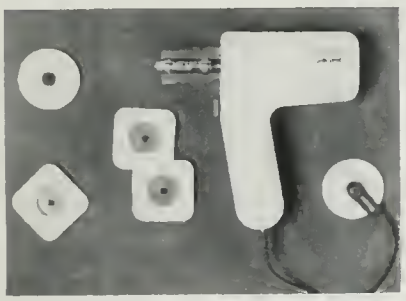

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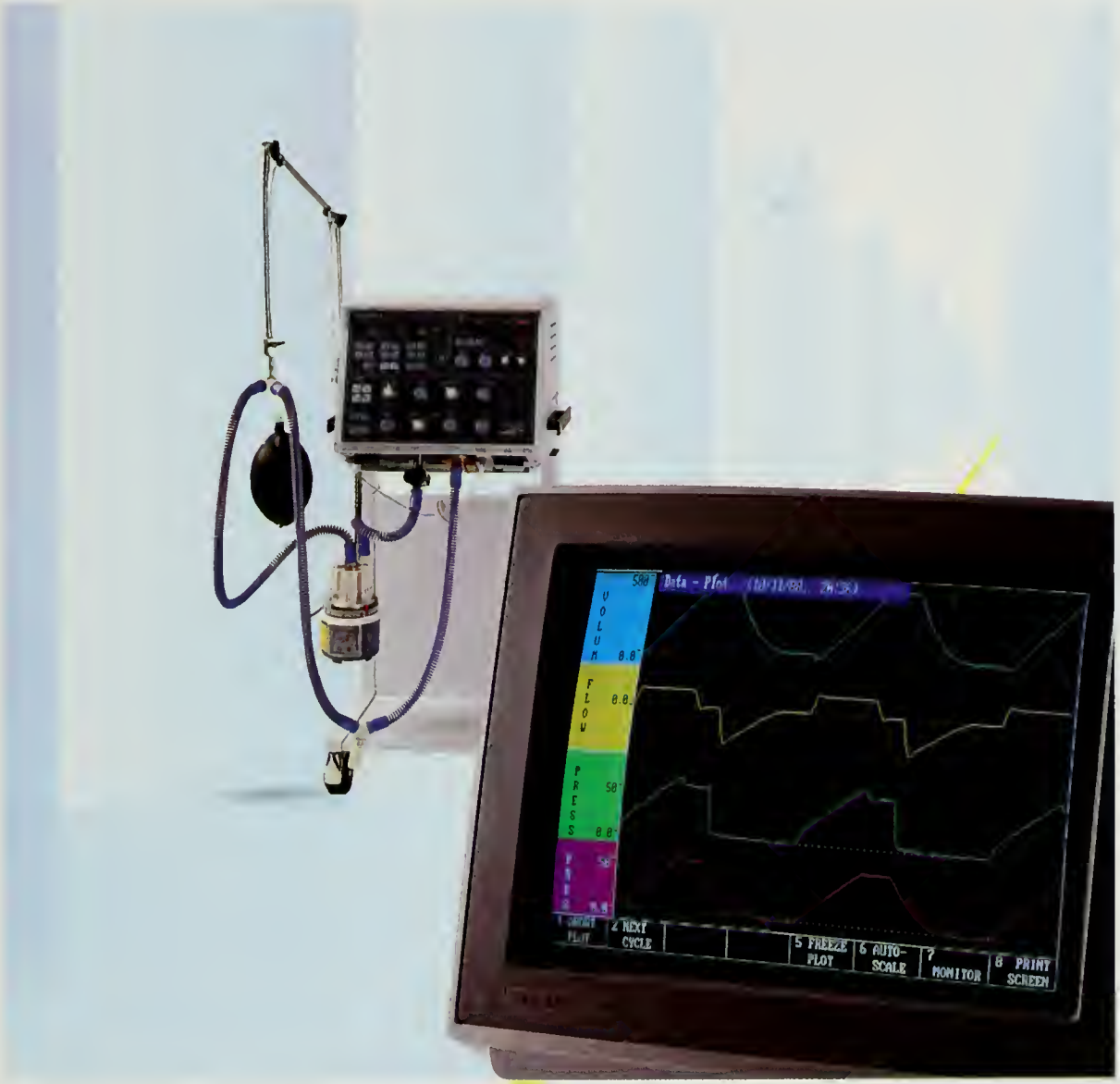

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